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THE HENRY JACOB BIGELOW LECTURE

SOME EXPERIENCES IN THE SURGERY OF THE OESOPHAGUS*

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TO come to Boston to lecture on the Surgery of the Oesophagus is like carrying coals to Newcastle, and I have much hesitation in addressing you on the subject. At the same time, it is of great interest and ought to occupy a more important place than it does at present. In the time of Henry Jacob Bigelow oesophageal surgery, as we know it, did not exist, but a great deal was familiar, many of your surgeons have been recognized as authorities, and your Dr. David W. Cheever is quoted in most of the books of the day for his success in oesophagotomy. Probably the knowledge of symptomatology and morbid anatomy was as full and as accurate as it is now.

"We think our fathers fools so wise we grow
Our wiser sons will no doubt think us so."

But the more we study the records of those old days, the more we come to admire their clinical acumen and the accuracy of their observations. Through the kindness of Dr. Hurst, of Guy's Hospital, my attention has just been directed to an account of one of the earliest cases of cardiospasm on record. The reference in the *Pharmaceutica Rationalis* of Thomas Willis was unearthed by Dr. Ralph H. Major of the University of Kansas. In this great work published so long ago as 1674, both the idea of cardiospasm or achalasia and the modern treatment are foreshadowed.

"No less will a very rare case of a certain man of *Oxford* show, an almost perpetual vomiting to be stirred up by the shutting up of the left orifice. A strong man, and otherwise healthful enough, labouring for a long time with often vomiting, he was wont, very often, though not always presently to cast up whatsoever he had eaten. At length the disease, having overcome all remedies, he was brought into that condition, that growing hungry he would eat until the *oesophagus* was filled up to the throat, in the meantime nothing sliding down into the ventricles, he cast up raw (or crude) whatsoever he had taken in: when that no medicines could help and he languished away for hunger, and every day was in danger of death, I prepared an instrument for him like a rod, of a whale bone, with a little round button of sponge fixed to the top of it; the sick man having taken down meat and drink into his throat, presently putting this down in the *oesophagus*, he did thrust down into the ventricle, its orifice having opened, the food which otherwise would have come back again; and by this means he hath daily taken his sustenance for fifteen years and doth yet use the same machine, and is yet alive, and well, who would otherwise perish for want of food. Without doubt in this case the mouth of the stomach being always closed, either by a tumour or Palsie, nothing could be admitted into the ventricle unless it were violently opened."

So far as my own experience goes, I can well remember the incident which led up to our recognition of the fact that simple strictures of the oesophagus might occur and lead to the death of the patient. On one occasion, as long ago as 1898, a man died suddenly after coming into the hospital with a history that for months he had been unable to swallow enough to support life in comfort. The diagnosis was of cancer, but a hurried postmortem, which had to be conducted in the coffin, provided us with the most complete and unusual specimen of simple stricture of the oesophagus that I have ever seen. The recollection of these early experiences reminds us that in the investigation of oesophageal cases, the profession owes a great debt to Roentgen, who has enabled us to see into the remote recesses of the body with wonderful exactitude. My own introduction to what one might call the aggressive surgery of the oesophagus dates from a second visit to Vienna in 1912. At that time we were being shown through the clinic by Professor Hochenegg when that great surgeon quite casually drew our attention to a case in which one of his assistants

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had made an anastomosis between the oesophagus and the cardiac end of the stomach, employing the transthoracic route. (Fig. 1 a & b.) At once the possibilities of this method flashed before me, and I longed for an opportunity of trying the operation. Principally owing to the distractions of the World War, no occasion presented itself until 1919, when a young man of nineteen came under my observation, who had complained for two and a half years of an increasing difficulty in swallowing. All kinds of treatment had been meted out with but little success, and at one period he had become so ill that gastrostomy was necessary to ward off impending starvation. After that, bougie treatment of various sorts was again carried out, but with only incomplete and temporary relief. After some further time the poor lad came to me in great distress, begging for something to be done, for he had become an outcast and was looked upon as a nuisance by his family, who grudged him food as he was too feeble to work. With such a free hand, I had no hesitation in carrying out the operation with which my Vienna visit had inspired me. It was difficult and trying, and convalescence was attended by much anxiety, but the lad eventually recovered and was able to swallow sufficiently to be independent of any other means. He took ordinary food with the rest of the family, returned to work, became a self-supporting citizen and was contemplating matrimony when unhappily, just two and a half years after the operation, he was knocked down by a motor car and received injuries which proved fatal. By the merest chance I heard of his death and was able to secure the specimen at a postmortem. The picture (Fig. 2), shows the anastomotic opening and the original stricture which, as you see, was not of the type we usually associate with cardiospasm, and which was so small that it would only admit the glass rod which has been placed in the specimen. Although this method of approach will probably be entirely superseded by the abdominal route, which I have myself employed in all my other cases, still it may have some uses not at the moment obvious, and it is worth mentioning the difficulties met with on that occasion. The greatest obstacles were the depth of the oesophagus from the parietes and the embarrassment caused by the incompletely collapsed lung. It appears to me that the first is insurmountable and it might be much worse than in my patient who was thin and small chested. To appreciate this difficulty, one ought to study the problem in transverse sections of the whole body. A preliminary collapse of the lung would do much to overcome the second trouble. The difficulties might be further lightened by employing combined or local anaesthesia, as in the wonderful series of cases of pneumonectomy carried out by Dr. Shelton in Toronto. In all such cases, whether carried out from the chest or by the abdomen, it would probably greatly facilitate the proceeding if we could temporarily paralyze the phrenic nerve, as mentioned by Dr. Harrington this morning. At the same time I must say that I have not found the intact diaphragm an embarrassment in dealing with the non-malignant cases which I am about to record. In all forms of interference in which the oesophagus is to be opened, it should be emptied by suction just before the operation and for similar reasons the stomach should be emptied and washed out through the gastrostomy. In any

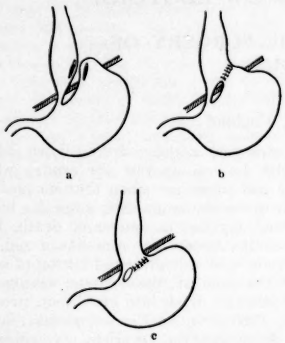

FIGURE 1. Diagrams of anastomosis between oesophagus and cardiac end of the stomach. (a) and (b) above the diaphragm, transthoracic route, (c) below diaphragm, abdominal route.

FIGURE 2. Showing the anastomosis made between the dilated oesophagus and the fundus of the stomach in the case mentioned in the text. (From Encouragements in Cancer Surgery—Grey Turner.)

event I was so impressed with the difficulties in this case that I looked about for some other method. At about this time I happened to come across a paper by Adrien V. S. Lambert of New York City (*Surg. Gyn. & Obstetrics* Jan. 1914), and it is only due to the well-known modesty of you American surgeons that I am unable to show you his photograph this evening. He advocated an abdominal approach to the lower end of the oesophagus, facilitated by separation of the left lobe of the liver from its diaphragmatic connections, a step which I have found a most admirable aid to exposure. After cutting through the peritoneum covering the abdominal oesophagus, it is easy by blunt dissection with the finger to mobolise that structure in the cellular plane in which it lies, and to draw it down into the abdomen sufficiently to enable the lower end to be inspected and explored and if necessary an anastomosis to be made between it and the stomach. This I have done several times and I want to tell you something of the results. Before doing so let me mention a few points in the technique.

This seems a proper place to mention that in nearly all the cases where a direct operation on the oesophagus is contemplated there should be a preliminary gastrostomy. For the most part this will be necessary to get the patient into a sufficiently good condition to tolerate the major interference, but quite apart from this consideration I believe it has great and obvious advantages in the after-treatment. I have always operated under general anaesthesia with the patient in the reverse Trendelenburg position, and have employed a long median incision commencing in the angle between the left border of the ensiform and the left costal margin and carried down to, or a little below, the umbilicus. (Fig. 3.) A long oblique incision parallel to and just below the left costal margin has also been recommended but I have not myself employed it. The upward extension of the vertical incision is the most important feature of this approach. It is of interest to mention in passing that as the extreme upper end is deepened the anterior muscular fibres of the diaphragm are exposed just in front of its central tendon. If these are incised, the pericardium is reached and may be easily opened. In one case this was done inadvertently but the opening was closed at once and no harm followed. I believe this would be the best and easiest route for deliberate opening of the pericardium for exploration or drainage. The next step is to gently take hold of the left lobe of the liver, which is drawn downwards, thus exposing the left lateral ligament. The latter is always very well defined and has an especially sharp left margin. (Fig. 4.) This ligament is divided with scissors along its centre and for its whole extent. By this manoeuvre, the left lobe can be completely mobolised and may then be either lifted out of the incision and held over to the right by some type of improvised retractor as shown, or it can sometimes as conveniently be packed away under the right side of the abdominal wall. This having been accomplished, the stomach is to be drawn well down when an excellent view of the intra-ab-

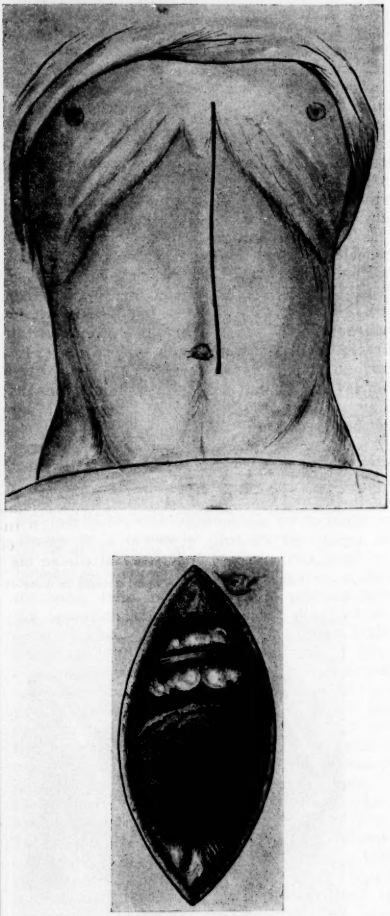

FIGURE 3. The incision recommended for abdominal exploration of the oesophagus. The smaller figure shows the exposure of the anterior fibres of the diaphragm and the way in which the left lobe of the liver hides the oesophago-gastric junction.

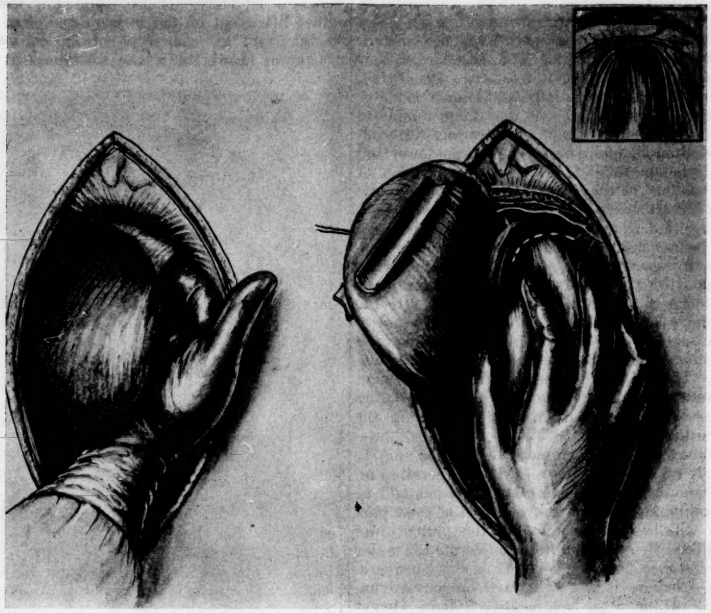

FIGURE 4. The separation of the left lobe of the liver from the diaphragm. On the right the liver is shown held aside by an improvised tractor. The dotted line indicates where the peritoneum of the abdominal oesophagus should be incised. Note the vessel running in front of the diaphragmatic orifice.

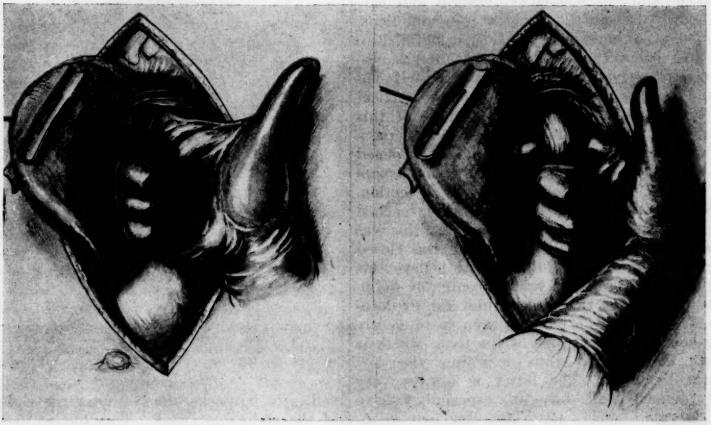

FIGURE 5. The mobilisation of the oesophagus from its cellular bed. On the right the oesophagus is shown so far loosened that it can be pulled down towards the abdomen.

dominal oesophagus, covered by peritoneum, will be obtained. It is now necessary to divide the peritoneum transversely at the point where that membrane is reflected from the diaphragm onto the oesophagus. A vessel which is always found running transversely in front of the oesophageal opening must be carefully avoided. The finger can now be introduced into the hiatus, and can easily be swept round the oesophagus which can then readily be separated from its cellular bed up to the highest point which the finger can reach. With the finger hooked round the tube, traction can be made on the latter, and it can usually be pulled down into the abdomen to an appreciable extent (Fig. 5), but unless it is elongated as the result of obstruction or is of the sinuous type, it is not usually possible to expose more than 2 to 2½ inches in this way. The lower oesophagus is now available for inspection and investigation. If it is decided to approximate it to the stomach, that can now easily be done towards the left as shown in the illustration (Fig. 6) and when an anastomosis is contemplated,

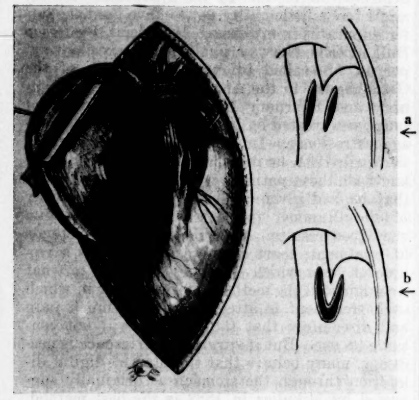

FIGURE 6. The first stages of the oesophago-gastric anastomosis. This may be made like Finney's pyloroplasty as at b.

a posterior layer of interrupted or continuous sutures is applied between the bare muscular wall of the oesophagus and the peritoneum-covered stomach. The oesophagus is usually very friable and stitches easily cut out and for that reason they must not be drawn tight. The opening in the oesophagus is to be made obliquely, or both it and the stomach may be incised after the manner of the Finney pyloroplasty. (Fig. 6b.) The oesophageal wall is much thicker than the stomach and therefore it is sutured to the muscular wall of the latter independently of the mucous membrane. It is usually possible to incise the muscular walls of both oesophagus and stomach without opening into the lumen of either. The mucous membrane of the oesophagus is very lax and bulges between the edges of the cut muscle. It must not be incised until the posterior suture line is completed. It is when the mucous membrane is incised that an accumulation of fluid in the gullet may be so troublesome; it may be removed by the sucker or a small gauze mop may be packed into the opening and left in situ, but it is much better to have the oesophagus previously emptied. It is important to make the incision of the mucous membrane of sufficient size and the opening should not be less than an inch and a half in length. The suture uniting the mucous membrane of the stomach and oesophagus must be continuous and of catgut and anteriorly it may include the deeper layer of the muscular coats. The anterior external sutures must also take a good hold of the muscle and are better interrupted. Sutures across the muscle fibres of the oesophagus are less likely to cut out than those placed vertically. It is important that one or two stitches should be placed at either end beyond the anastomosis. I have usually put a couple of stitches through the anterior margin of the oesophageal opening of the diaphragm to fix it to that tube above the anastomosis so that the latter is confined to the abdomen. The left lobe of the liver is then fixed to the stomach just below the anastomosis by a mattress stitch. This not only protects the union by acting like a graft, but is also a means of anchoring the mobilised lobe. As a matter of fact the latter falls comfortably into position over the operation area and probably does not require fixation of any sort. If there is any doubt about the security of the suture line, I have no hesitation in bringing a small rubber drainage tube from the region out through the parietal incision. I feel sure that in my last case of successful total gastrectomy, this was the means of saving the life of the patient. No special after-treatment seems necessary. Most of my cases have been operated upon in the presence of gastrostomy, so that feeding could be carried out entirely by that route. In its absence the patients have been allowed to have sips of water from the first, and if that can be swallowed without discomfort, liquid nourishment continues to be administered in that way. Otherwise for the first few days rectal alimentation must be resorted to. None of the cases have given any cause for anxiety during convalescence and there have been no special complications.

The first case in which I had an opportunity of using this method was that of a young woman of twenty-one, who came under my observation in 1923. She had been troubled with difficulty

in swallowing since the age of eight and had been treated in and out of hospital by many methods. At the age of fifteen she was taught to use a bougie. As a result she was a little relieved, but it caused her great pain and was soon given up. Her difficulty increased, she became very miserable, was constantly regurgitating mouthfuls and had to sleep in a sitting-up position. The x-ray showed the typical picture of cardiospasm. In her unsuccessful efforts to obtain relief she had attended the outpatient department of the same hospital at which the previous patient had sought treatment, and she was so delighted at the result in his case that she begged, nay demanded, that the same operation should be carried out for her. It was not possible to persuade her to try any other sort of treatment, nor would her parents consent, and it was only against my better judgment that I agreed to operate. Exploration of the oesophagus did not disclose any naked eye evidence of any pathological condition and under the anaesthetic a large bougie could be passed into the stomach with ease. Had I not believed that there would never be another opportunity of interfering in this case, I would not have felt justified in doing anything further but as it was the anastomosis, as I have described it, was carried out without much difficulty. She made a splendid recovery, subsequently married, became the mother of two healthy children, and now, eight years after the operation, is well and happy and without any suggestion of recurrence of her old trouble. But I do not wish you to think that I have come here to advocate this plan rather than the other means available, nor do I make any bizarre claims for its adoption. I am well aware of the excellent work that has been done in your country by the various methods of dilatation which have been employed, and we always endeavour to treat cases in that way in the first instance. With us the favourite method is the use of the mercury bougie, introduced by Dr. Hurst of Guy's Hospital, and in many cases it has proved wonderfully successful. The following is Dr. Hurst's own description of his method (from Achalasia of the Cardia [so-called Cardiospasm.] *Quarterly Journal of Medicine of Great Britain*, July 1930). "As it is presumably impossible to restore the power of relaxation to the closed cardiac sphincter, the object of treatment must be to dilate it to such an extent that it no longer offers any resistance to the passage of food into the stomach. This can be done most easily and with least discomfort and danger by means of the mercury bougies which were first described by Hurst in 1913. Rubber tubes varying in diameter between 33/64 and 46/64 inch (gauge Nos. 28 to 34), 31 inches long, and each containing the same weight of mercury (1 lb. 5 ozs.) which has been found by experience to be sufficient to force the closed sphincter, are now obtainable. It is best to pass the bougies on the first occasion during an x-ray examination; it is then seen that they pass without deflection straight into the stomach. A mark should be made on the bougie at the level of the teeth when its two lower inches are within the stomach. Successively larger tubes are passed at a single sitting, and in most cases the largest one meets with as little resistance as the smallest. The patient is then taught to pass the largest bougie on himself before each meal. He finds at once that a meal taken after its passage enters his stomach without difficulty. For a few days the bougie should be retained in position on each occasion for as nearly five minutes as possible; then the time is reduced before lunch and dinner, and after within a week the midday passage can be given up entirely. Later the bougie is passed in the morning only, and the time it is kept down is steadily reduced. After a time in favourable cases the bougie is only passed on alternate days, then once a week, and finally its use is discontinued, but it is kept by the patient so that he can pass it if at any time he feels that some slight obstruction has returned." At the same time I am not at all sure that we have not neglected the method by the hydrostatic dilator, and I feel a little guilty not to have used the latter apparatus in my cases. My friend, Professor Bull, of Oslo, Norway, published an excellent paper on "So-called Idiopathic Dilatation of the Oesophagus" in the Annals of Surgery for January and February 1925. He there recorded four cases treated by infradiaphragmatic oesophagogastrostomy. In response to my enquiry in April, 1931, he informed me that so far as he knew all those patients were going on well, but that he had given up the operation in favour of the Plummer probe which had given him very good results. Nevertheless, by all methods of treatment, short of operation, there is a residue of cases which either do not yield to what we might call the methods of election or in which such treatment is attended with so much pain and discomfort that the patients will not continue its use. But if surgical interference is necessary, many believe that the plan of digital dilatation through the stomach as originally suggested by Mikulicz in 1882 is the best method. My friend Mr. A. James Walton, of London, has treated twenty-two cases in this way; in 14 of these cases prolonged treatment by bougies had failed. In three cases death followed, but in thirteen out of nineteen the patients have been permanently cured over a period of many years. (The Neuro-Muscular Obstructions of the Gastro-Intestinal Tract. London. *Lancet*, December 20, 1930.)

It is somewhat of a delight to the general surgeon to be called upon to deal with the occasional failures emanating from the clinics presided over by specialists, and so it is with these oesophageal cases!

Let me tell you of some other of my experiences in this field. The French surgeons have

held that in cardiospasm it suffices to expose the oesophagus from below, to loosen it from its cellular bed, and to draw it down towards the abdomen. This is a plan which has been carried out in five of my patients. It is most striking to be confronted with a typical case of cardiospasm which has resisted treatment by dilatation and other conservative means and which, after exposure in this way, shows no evidence whatever of such gross pathology as can be detected by the naked eye. After this loosening up process it is often possible to pass the largest size bougie without the least difficulty and to have ocular proof that it passes directly into the stomach. It was because of the absence of a demonstrable lesion and the ease with which a large bougie could be passed on the operating table in my cases that I felt that no plastic operation or anastomosis seemed indicated or justified. However, the results have been illuminating because only one patient, a female, operated upon six years ago has attained anything like permanent relief.

Many surgeons have looked upon the condition of cardiospasm as allied to the stenosis of the pylorus of infants, and attempts have been made to treat it on the same principle, sometimes with results apparently permanent. R. P. Rowlands, of Guy's Hospital, treated one of Dr. Hurst's patients in this way and the patient remained well four years later. I have had two cases which were operated upon by this method (Rammstedt); that is thoroughly exposing the oesophagus, cutting through the muscular wall vertically until the mucous membrane is exposed and leaving the muscle unsutured. Both these cases recurred, and at the second operation there was more scar tissue and much more trouble in again exposing the oesophagus than if nothing had been attempted. One such case was a boy (Greener) nine years of age who gave a long previous history of difficulty in swallowing, treated by medical means and by bougies, and eventually demanding gastrostomy. In September, 1922, the Rammstedt operation was carried out, but he got relief for only a month, when his attacks continued as before. In February, 1927, the abdomen was re-opened and an anastomosis made after the method already described. Three weeks later he wrote on his bed card "This is the first time I have eaten properly for eight years." He got so well that he was able to go to sea as an apprentice, and when I saw him after one of his earlier voyages he told me that he could eat ordinary food and that he could eat it quite quickly, for, as he said, if he did not do so, the other apprentices might get more than their share! That was four years ago and although he has at times some little difficulty, he looks upon the result as a brilliant success.

The next patient was a man (Coulson) forty-one years of age, who came to the hospital complaining of vomiting of twelve months' duration and who had been treated in the medical wards as a functional case. In March, 1927, he was admitted to the surgical side, where the abdomen was explored. The stomach and gall bladder were found to be normal, but of course the appendix was removed. During convalescence the real nature of his complaint was discovered and treatment commenced with Hurst's bougie. For a time he did extremely well, put on weight and was fairly content, but after two years the condition recurred in spite of the continued use of the bougie. He had much pain with regurgitation and rapidly lost weight and it was obvious that something further would be necessary.

The x-ray picture was very typical of cardiospasm and showed dilatation higher in the oesophagus than before. In September, 1929, I made the abdominal anastomosis. The result was immediately successful and he gained ten pounds in three weeks. Now, twenty-one months later, he is keeping well and is able to take ordinary food without inconvenience.

The next patient (Osborne) to whom I wish to refer, has had a long and chequered history. He is a member of the industrial class whose illness began in 1919 with what he called indigestion accompanied by some interference with the act of swallowing. From that time he had been under many doctors and had been treated in various ways. Eventually the abdomen was opened, and as the stomach and gall bladder appeared normal, the appendix was removed. As he was no better all his teeth were extracted in the hope of improvement. Finally in October, 1924, a diagnosis of cardiospasm was made, and another surgeon explored the lower end of the oesophagus. Nothing abnormal was discovered, but gastrostomy was carried out. After a time he became able to swallow a little better, but this improvement was only intermittent and for the most part he had to depend on the gastrostomy. Sometimes during an attack of difficulty he was so bad that he could not swallow even saliva. This man first came under my own personal observation in July of 1928. He was then 33 years of age, thin, anaemic and ill looking, and told me that for nearly 3 years, he had depended almost entirely on feeding by the gastrostomy. During this time his weight had been as low as 98 pounds. The x-ray finding was typical of cardiospasm. Treatment by Hurst's bougie was commenced at once and with great relief, for he began to take more or less ordinary food with ease and satisfaction. He learnt to use the bougie himself, and passed it daily. After three months, for no obvious reason, the passage of the bougie was followed by great pain and he had to discontinue its use and went rapidly downhill. In that month, i.e., October, 1928, the oesophagus was explored from the abdomen and thoroughly loosened up from its bed, the muscular wall being exposed over a considerable

length. As no pathological condition could be demonstrated, and a large bougie could be easily passed, nothing further was done. For a month he got considerable but not complete relief, and both he and I were much disappointed. However, he was able to resume the use of the bougie which he carried on for the next twelve months. In October, 1929, he was so uncomfortable that he was content to risk a further operation in the hope of permanent relief and I repeated the exposure of the oesophagus as before. This time the left lobe of the liver was buried in dense adhesions from which it had to be literally dug out and was very considerably torn, leading to a good deal of haemorrhage. The oesophagus was also very much hidden by adhesions and was difficult to expose. Eventually a satisfactory anastomosis was made but I took the precaution to reëstablish the gastrostomy which had been allowed to close. Recovery was satisfactory and rapid, and in about a fortnight he was able to take light food by the mouth. The gastrostomy was soon allowed to close and early the next year he managed to get employment as a labourer and was able to work as hard as before. In May, 1931, he came to report, just 19 months after the last operation. He stated that he was perfectly well and had gained much weight, so that he was then 144 pounds. He could eat any sort of ordinary food like a normal person, but he had to take a little more time than his fellows and could not "gulp down his meals". I was very anxious to have some x-ray confirmation of the functional capacity of the anastomosis which this happy state of affairs seemed to portend and so the patient was sent down for examination after an opaque meal. To my great chagrin the screen examination showed that the meal was held up for 25 minutes before it passed into the stomach, and that it then appeared to do so by the original oesophageal opening. A film showed the typical appearances of cardiospasm of moderate degree. This was just before I left home and there was not time to arrange about an examination by the oesophagoscope. I sometimes wonder if the nasty bismuth porridge, which we use for these investigations, is as likely to disclose the normal condition as some more palatable meal which the patient desires. In another case in which the x-ray finding was not in keeping with the patient's story of the ease with which he could take food, the porridge was held up for a considerable time and seemed reluctant to pass into the stomach, despite the fact that the patient was standing. One of the x-ray attendants, a man with considerable experience, said that he had noticed a similar reluctance in apparently normal persons. This observation prompted experiment and to show a good example I stood before the screen and took some mouthfuls of the unappetising meal which dropped like lead into my stomach without the slightest hesitation. My house surgeon was then examined, but in his case the opaque porridge behaved pretty much as it had done in the patient! The only difference of which I was aware between my house surgeon and myself was his fortunate youth and the fact that he had also the advantage of being a Scotchman!

In all but the first case so far mentioned the diagnosis of cardiospasm or achalasia was accepted and they were dealt with on that assumption. The next case to be referred to appeared to be of a different type and at least when finally dealt with by me presented a simple cicatricial stenosis of the lower end of the oesophagus. The patient (Fulham) was healthy in infancy and early childhood, but at the age of 8½ began to vomit immediately after taking food. There was no pain but he rapidly wasted and his state caused much alarm. At this stage he was treated by physicians and was kept in hospital on more than one occasion and for various periods, but with only temporary benefit. In August, 1924, when the boy was just over 10, one of my colleagues exposed the lower end of the oesophagus and carried out Rammstedt's operation on its extremity. The boy made an easy recovery and was much better during the few weeks he remained in the hospital, being able to take light food by the mouth, but as soon as he returned to his home he began to vomit as before and steadily lost ground. By this time both the boy and his parents were naturally very disappointed and became so despondent that they would not allow the patient to return for treatment. Finally the condition became desperate; he was scarcely able to swallow at all; he had lost weight seriously and had become intensely anaemic and generally oedematous—closely resembling the condition seen in beriberi. In this state he was finally re-admitted to hospital in March, 1926, being 12½ years old. His condition was then alarming and I have never seen anybody so starved. After a short preparation I carried out gastrostomy using local anaesthesia. The boy rapidly improved and in two months it was considered that he was well enough to justify a further operation on the oesophagus. This time a definite cicatricial stenosis of the lower end was disclosed and this was treated by cardioplasty—the old-fashioned Heineke-Mikulicz pyloroplasty carried out at the oesophageal end of the stomach (Fig. 7). The vertical incision was 1½ inches long and was carried into the lumen throughout its length. Recovery was rapid and complete, and in May, 1931, just 5 years after the operation, he is well and strong and working regularly and writes "I take normal food like a normal person."

With regard to this plastic manoeuvre I have had an extensive experience of this type of operation for pyloric stenosis, and I know that when the lesion is really a cicatricial stenosis without active ulceration it gives most wonderful results.

No patients make more rapid or complete recoveries, and I have known them actually to double their weight in the first few months. It is only the march of surgical progress which has rendered the indications for pyloroplasty so rare, for patients demand relief in every sense of that word, long before the stage at which cicatricial stenosis occurs. Judging from this experience

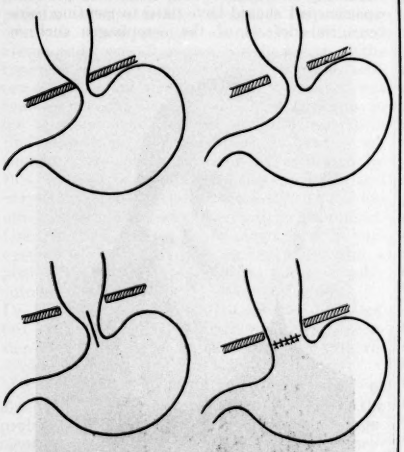

FIGURE 7. Cardioplasty, the steps of the operation as carried out in the case (Fulham) described in the text.

with the pylorus I should expect that a similar measure would be equally satisfactory at the cardiac end of the oesophagus. Years ago I had a case of cicatricial stenosis at the junction of the oesophagus with the stomach resulting from a healed ulcer in the latter which might have been admirably treated by this method of operation. Our Museums display only a few specimens of this nature, but the one shown in Fig. 8 is a typical example from St. Bartholomew's which I think could have been easily and permanently cured by cardioplasty. But strictures of cicatricial origin at the lower end of the oesophagus are extremely rare (at least in England) and the greater number of obstructions which present themselves are of the nature of cardiospasm which is undoubtedly of neuromuscular origin. For this condition there can be no doubt that some type of dilatation is the method of choice and experience shows that it is apparently never too late to carry out this treatment with hope of success. A striking illustration came under my observation in the person of a married woman in the fifties (Carr). In January, 1911, gastrostomy was carried out for what was looked upon as an irremediable stenosis of the oesophagus. In July, 1915, she turned so tired of depending on the gastrostomy that an attempt was made to deal with the stenosis, using swallowed silk thread as a guide for a bougie: this was successful and she resumed normal feeding and allowed the gastrostomy to close. Nevertheless in October, 1927, that is 17 years from the onset of her trouble, this patient still suffered from occasional attacks of spasm causing typical symptoms for a few hours. However, personal experience, a study of the litera-

FIGURE 8. Cicatricial structure at the lower end of the oesophagus (a specimen in St. Bartholomew's Museum. Copied by permission).

ture and equally important information gleaned from friends shows that there is a residue of cases in which treatment by conservative means has not been permanently successful. In such cases I have no hesitation in recommending the exposure of the oesophagus, which I have described, followed either by cardioplasty or anastomosis whichever appears likely to fulfill the indications best. There are also cases where the oesophagus is so sinuous that it is almost impossible to guide a dilator or bougie into position, or others in which there is extensive ulceration likely to make bougie treatment very dangerous. These cases would appear to me to fur-

nish good grounds for contemplating the direct surgical interference described. Most of the examples I have recorded show how little these curious cases are understood and unhappily they do not appear to me to shed light on the pathology. Until the causation is discovered it seems to me that treatment must continue to be haphazard and empirical.

One of my colleagues, Mr. James Whillis of our Anatomical Department, is investigating the problem of the oesophagus and its functions, and he tells me that it is very difficult to arrive at any definite conclusions. On some points he has gleaned definite information; for instance in both man and the cat there is no evidence of a cardiac sphincter whereas in the rabbit there is a definite cardiac sphincter composed of striped muscle. In both man and cat the diaphragm obstructs the oesophagus, particularly during inspiration, and no other constricting mechanism is evident in the wall itself. In the cat the diaphragmatic fibres bounding the oesophageal opening go into spasm in deep anaesthesia but division of the phrenic nerve causes no obvious relaxation of such spasm. The musculature of the diaphragm and its relation to the oesophageal opening in man certainly suggests that it has a sphincteric action on that tube. On the other hand the oesophagus itself is only a hollow muscle. Observation in man, and experiments in the cat suggest that any abnormal condition of the oesophagus or its surroundings may cause spasm of its wall, but there is no doubt this spasm is most likely to occur at the spot where it traverses the diaphragm. Hurst takes the view that the lesion is not a spasm but an incoördination of the mechanism of relaxation which he suggests is due to a degeneration in the ganglion cells of the nerve endings of the vagus in its wall, and Professor Adrian Stokes demonstrated this condition in one of my own cases. Whatever the explanation, it is not easy to see how the condition can be relieved by making such an anastomosis as I have described unless there is some continued obstruction at its lower end. It is significant that the disease was not cured in five out of the six cases in which the oesophagus was only thoroughly loosened up from the diaphragm.

Let me just mention some of that group of cases in children which occur in tender years but which are apparently not developmental and in which the obstruction occurs between the crossing of the left bronchus and the diaphragm (level of 7th thoracic vertebrae).

One of my physician colleagues (A. G. Ogilvie, in collaboration with Wilfred Sheldon. *Archives of Disease in Childhood*, 1929, 4, XXIV, 347) has collected and closely observed a series of these cases and has deliberately come to the conclusion that the majority get spontaneously well without any treatment other than restriction of diet to easily swallowed fluids. A remarkable example of this sort came under my own observation in the person of a child who had signs of obstruction from birth. At the age of three indefinite stenosis just above the diaphragm was demonstrated by x-ray. At the age of six the trouble seemed suddenly and spontaneously to disappear and at eleven the child was practically normal.

Had my address not been limited to personal experiences I should have liked to mention those congenital defects of the oesophagus such as

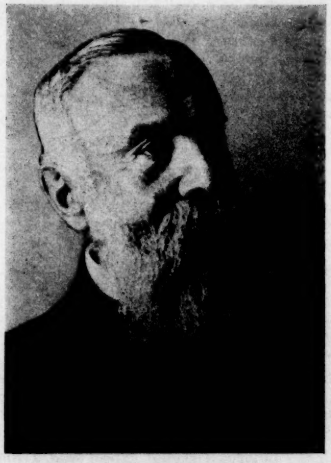

FIGURE 9.

atresia, congenital stricture and oesophago-bronchial fistula which should not be accepted as entirely incurable. Strictures due to the swallowing of caustic fluids are now very rare in Britain, but in the year 1912 they were common enough among children in Vienna. I have the most vivid recollection of seeing a whole series of these social calamities under treatment by Professor Lotheissen. The children were being taught to pass their own bougies and it was indeed an extraordinary sight to see a row of little ones of tender years sitting side by side on a high shelf and demonstrating their capacity as oesophageal surgeons under the persuasive hypnotism of the painstaking Professor!

Let us now turn to what has usually been considered the most unsatisfactory subject in the whole realm of Surgery—Carcinoma of the Oesophagus. In most clinics this tragic condition is treated with an almost studied neglect or an attitude of benevolent despair. Surely such

a state is a reproach to Surgery and cannot be left where it is. In Britain there were no less than 15,909 deaths attributed to carcinoma of the oesophagus during the ten years 1911 to 1920 (Souttar). With a path strewn with disappointments we might be excused for leaving the surgery where it is if other methods held out hope of relief, but diathermy, radium and x-ray therapy are not fulfilling their earlier promise or expectation. I cannot help feeling that the whole subject should be considered afresh and from all angles. Surely a man of the type of Henry J. Bigelow (Fig. 9), in whose memory the beautiful medal I have just received was founded, would have tackled this problem in his intensive way resolved and determined to find a solution. The textbooks, at least in my country, are not encouraging as witnessed by this extract from one published in 1927, and very widely used. "Oesophagectomy. This is another operation introduced only to be abandoned. Czerny's case, it is true, was temporarily successful, the patient living rather more than a year after the operation. But cases equally suitable . . . must be quite exceptionable . . . De Quervain has collected a number of cases, but the results are not encouraging. The operation mortality is heavy, and in those who recover prolongation of life is unlikely."

What statement could be more likely to deter anyone from even considering the problem? I prefer to remember the encouragement I derived from the conversation of Theodore Kocher, who did so much to arouse interest in the operative treatment of malignant disease. He certainly recognised that hope and encouragement are essential to stimulate the best effort.

> "Work without hope draws nectar in a sieve
> And hope without an object cannot live."
> *Coleridge.*

I do not understand why you in America have not been more enthusiastic about the really brilliant achievement of your countryman, Franz Torek of New York City. His case of Oesophagectomy carried out by the posterior mediastinal route was a wonderful success, for despite the fact that the fistula in the neck and the gastric fistula were never united and that the gap had to be bridged by a rubber tube, the patient lived in comfort for nearly 14 years and died at the age of 80 without recurrence!

In approaching this problem most workers have assumed that the disease universally progresses rapidly and early disseminates, but I venture to suggest that this is not in accordance with the facts. Thirty years ago the following case came under my observation and has remained an abiding encouragement. In those far-away days, x-rays, so far as the oesophagus was concerned, had not become part of our diagnostic armamentarium, or the confusion associated with the diagnosis would not have occurred. The patient was a man aged 52 who was admitted to the Newcastle Infirmary in 1901 suffering from oesophageal obstruction of 2½ months' duration, and demanding gastrostomy. After that operation he improved so much that in a few months he allowed the opening to close, but his symptoms soon recurred, and the operation had to be repeated. Again improvement was so complete that the original diagnosis of cancer was doubted, and antispecific treatment was carried out. As this did nothing to restore his swallowing capacity we endeavoured by the use of bougies to overcome what we looked upon as a simple stricture. This was only partially successful so the stomach was opened and by the retrograde method it was found possible to pass a good-sized tube through the stenosis. This was removed at the end of a week when he found himself able to swallow comfortably. In due course we assayed to pass bougies by the mouth to maintain the improvement, but this was found impossible and in order to provide against recurrence I was deputed to make a further effort under anaesthesia. There was great difficulty but just when we were about to give up the attempt the bougies slipped through and everyone was delighted. But the man never picked up, he developed left-sided pleurisy with effusion and died in a week. At a postmortem we found that the liquid food taken by the mouth had passed straight into his left chest, and there was extensive pleurisy with collapse of the lung. The lower third of the oesophagus was occupied by a long linear typically carcinomatous growth. Though death occurred in this way one year and seven months after the first operation, or 21½ months after the first symptoms there was no evidence of local or general dissemination. This one case alone led me to believe that cancer of the oesophagus was not always the rapidly fatal disease reputed, and that some day it might be amenable to operative interference. Many careful observers have had similar experiences and I venture to say that while 50 per cent. of cases may be of the type that early spreads an equal number remain localised for long and though they may do irreparable damage by continuity of tissue they do not spread to the glands or liver. If we are to do much good in these sad cases, there must be a change of heart in the profession, and I am prepared to admit that this may be more necessary in my country than in yours. So far as my observations go, your people not only apply for relief earlier than ours, but are more willing to submit to more rigorous examination in the hope that it may be followed by effective measures for relief.

In the immediate past, if not at present, when a patient complains of difficulty or hesitation in swallowing, there is a tendency to temporise until such time that there is difficulty in swallowing enough nourishment to satisfy hunger when the question of gastrostomy or intubation is considered. With the means at our disposal

this attitude is indefensible and any form of successful treatment must presuppose as early interference as possible.

The incidence of malignant disease in the oesophagus in its various sites is differently stated by various observers, but the following figures published by Souttar of the London Hospital accord fairly well with my own experience. He found that in 100 cases, 31 were situated in the upper end, 16 at the lower and 53 in the middle portion. I think there is some evidence to show that the type of growth varies in the different regions, but this is a matter that deserves much more careful investigation than it has received. At first sight it would appear that the growths which attack the upper end of the oesophagus are less malignant than those in other situations.

I must at once say that my own personal efforts in the excision of malignant growths of the upper end have not been attended with success, but I have had much encouragement, and greatly admire the success that has attended the efforts of some of my friends. On anatomical grounds, growths in this neighbourhood present a favourable opportunity for extirpation, especially if the circumstances of the cases, such as implication of the larynx, together with the courage of patient and surgeon face the very radical extirpation which the method of Gluck provides.

Proceedings less radical are attended with great difficulties in the restoration of the canal and it is not easy to see how they are to be overcome.

At first sight growths in the lower end of the tube would appear to be much more suited for extirpation, but the rigidity of the oesophagus or the absence of elongation in malignant cases makes it difficult at one and the same time to carry out a sufficiently wide extirpation and yet allow of approximation of the severed oesophagus to some portion of the stomach. At least these have been my difficulties and though I have twice carried out such an excision and made an anastomosis, neither of the patients survived. These operations were difficult, and in future I would certainly seek what help division of the left phrenic nerve might provide. Guided by my experience of total gastrectomy I would also advise that a small safety drain should always be brought from the neighbourhood of the anastomosis to provide against an unexpected leakage.

In dealing with growths in this situation I have found that they have a tendency to extend posteriorly more than laterally or anteriorly and when excision is contemplated it would be wise to determine the posterior relationship as an early step. In one case in which I excised the growth, the left suprarenal was infiltrated. A most encouraging case has been published by Professor E. Gohnbrandt. (*Langenbeck's Archives for Clinical Surgery* 148, 1927, pp. 528.) This surgeon successfully excised 5 cms. of the lower oesophagus, containing a carcinoma, and joined the upper end directly to the stomach. The patient made a good recovery and was able to swallow comfortably until death occurred six months later from embolism secondary to phlebitis of a lower limb. Postmortem examination showed that there was no recurrence of the growth and that the union was entirely satisfactory.

At first sight growths in the middle part of the oesophagus appear to present the most insurmountable problem. Despite the one brilliant success of Torek and of Eggers and the courageous efforts of many others, the external posterior thoracic route appears to me to present almost insurmountable difficulties. In pictures, with their facility of making things look easy, the method appears feasible enough, but even the most optimistic may well be deterred by a study of transverse sections of the whole thorax. Only those who have tried this plan can realise the depth and inaccessibility of the central part of the oesophagus. Even granted successful extirpation, the restoration of the canal is a problem of very great difficulty. A good many years ago it occurred to me that it might be possible to enucleate the oesophagus from its tunnel, working from the abdomen and the posterior triangle and withdrawing the whole tube. This was first suggested by a picture attributed to Torek which shows the greater part of the oesophagus hanging free on the front of the thorax from an incision in the posterior triangle. In the War time the Hospital to which I was attached was halted at Alexandria on its way to Mesopotamia. It was in those dark days of early 1916 when Townsend was being invested in Kut, and when all were anxiously waiting for the daily expected announcement of his relief. One day I noticed on a poster announcing the current issue of "*John Bull*" these startling words "Relieve Townsend, Remember Gordon." Instantly and for the first time the terrible thought flashed across my mind that perhaps after all it would be impossible to relieve the gallant General with his faithful defenders. And so it was that the picture of Torek's flashed across my mind the idea that the *whole* oesophagus might perhaps be enucleated from its bed leaving the problem of its substitution by other means. The idea developed in my mind and I began to look into the possibilities. Certain anatomical considerations required thought, for the oesophagus is intimately associated with the most important structures in the thorax, and a superficial examination certainly suggests that its removal by any means would be well-nigh impossible. Could the oesophagus be separated from its bed without direct exposure, what would happen to its nerves

if that were done and how about the vessels? So far as the first question is concerned, I had little doubt, for like other tubes such as the trachea, the ureter and the big arteries, that hollow muscle lies in a bed of cellular tissue with which it is very loosely connected, thus allowing for the variation in size and freedom of movement necessitated by the act of swallowing. There is some attachment by firm fibrous bands to the neighbourhood of the left bronchus, but elsewhere it lies free in its bed of cellular tissue except for the anchorage of its vessels. The vagi are spread out on the wall of the oesophagus and in any attempt at enucleation would certainly run the risk of being torn, but it has been shown, paradoxical though it seems, that both can be severed in the chest without serious consequences. The question of the blood supply appeared to be the crucial matter. Fortunately there are no large individual trunks, except those in the neck from the inferior thyroid, but numerous small branches from the intercostals, especially where those vessels come off from the aorta. Then one was familiar with the extraordinary development of the veins seen in cases of oesophageal varix and it appeared as though they would form a great obstacle, but as a matter of fact, except when pathologically engorged, they are not of great size. In my country it is not easy to get permission to experiment on animals, and it seemed that the only thing to do was to make observations on the human subject. With this in view, I supplemented work in the anatomy and postmortem rooms by exploration of the oesophagus from the abdomen in several patients. As a rule when these cases come to hospital, they are suffering from actual or incipient starvation and are not in a condition to tolerate any but the most necessary interference. I have found it best to bring relief by an immediate gastrostomy, and to postpone any but the most necessary diagnostic measures for two or three weeks, by which time their state of semistarvation has disappeared. For years now the inverted ink-bottle type of gastrostomy, known in my country as the Kader-Senn method, has been almost universally employed and in my experience, at least, it has given excellent results. But it is a curious fact that despite the amount of liquid nourishment that can be administered by this means, patients never improve so quickly or so completely as when mouth feeding can be employed. They also do better if they can swallow their saliva or if it is fed to them through the gastrostomy tube. If the patient does not show marked improvement as a result of the gastrostomy, this in itself is a bad prognostic sign and suggests, though it does not prove, that there are already secondary deposits. Before admitting such a conclusion, the element of sepsis should so far as possible be eliminated. For this purpose the best plan is to allow the patient to try and swallow frequently small quantities of a one in ten solution of peroxide of hydrogen (40 vols.). This by its ebullition removes discharge, loosens débris and food particles and generally greatly helps. If this does not improve matters then the oesophagus may be gently irrigated. Some surgeons have stated that these patients are greatly improved by extraction of all the teeth, but I have not found it so. If the improvement justifies further measures, then all possible information that may be gleaned from x-ray examination and oesophagoscopy should be obtained, but so far as my experience goes, all the diagnostic measures that can be employed stop short of providing the information that is most required, namely the amount of extension to the parts outside the oesophageal wall. A closer study of the relation of the length of history and the clinical symptoms to the pathological findings in a considerable group of cases might elucidate many of these points and is worthy of careful consideration. It may be wise to mention the necessity of looking for those signs which indicate that the case has already passed beyond the operable stage. This may be shown by the presence of secondary glands at the root of the neck on one or the other side, or on both; or paresis of the recurrent laryngeal nerve, or of the sympathetic. Or there may be detectable secondary deposits in the liver or evidence of extension to the root of the lung or the pleura. The absence of any of these signs must not be assumed because the patient looks in good condition, and makes no special complaint other than the original difficulty in swallowing. Having decided to carry out abdominal exploration, the following technique is employed. With the patient in the reverse Trendelenburg position, the same incision is used as that described in the earlier part of this paper. If this does not give sufficient room or if the parts are especially deep as in very stout people, further help may be obtained by cutting across the left rectus muscle at the junction of the upper with the middle third of the vertical incision, and in any case the oesophagus is exposed by separating the left lobe of the liver from the diaphragm. With the finger in the proper cellular plane—the oesophageal bursa—separation is carried out until the digit can be passed completely around the tube. (Fig. 5.) The forefinger is then worked upwards in the cellular plane until the growth is reached and is gently passed around it so that its connection can be made out. Meanwhile gentle traction may be made on the oesophagus to facilitate the task of the examining finger. Traction may be aided by passing a strand of gauze or a strong tape around the tube. I have now explored eight ex-

amples of malignant oesophagus in this way, but in all but one the growth had so far invaded the parts outside the tube as to render its removal impossible. The one case in which it was carried out will be described later. In another (Vipond), it seemed all but feasible, the patient being a thin man in his fifties and the growth situated about the crossing of the left bronchus. At the exploration I was able to reach the growth with the finger and to separate it all around except at one point where it

FIGURE 10. Malignant growth of oesophagus from case (Vipond) mentioned in the text.

appeared to be adherent to the bronchus. I was afraid to persevere lest the latter should be torn so that I was reluctantly obliged to abandon the attempt. The man was not greatly upset by the interference, but developed septic pneumonia from which he died on the fourth day. At the postmortem examination there was considerable blood staining of the left side of both the chest and abdomen. There was no evidence of sepsis either in the mediastinum or the lung. The left pleural cavity contained about ten ounces of blood and serum, the corresponding lung was partially collapsed and its lower lobe presented a large infarction about the size of a cricket ball. There was very little extrapleural bleeding. The growth (Fig. 10) could not be further separated by blunt dissection with the finger working from below. After exposure it was found to be involving the right pleura at one point and the wall of the aorta at another, but the latter was not firmly or deeply infiltrated. Nevertheless it was necessary to use the scissors to complete the separation at these points. Beyond this spread by direct continuity there was no glandular involvement and no distant dissemination and at an earlier stage the oesophagus could probably have been enucleated with ease.

I am sure it is a mistake to make the exploration unless the operator is prepared to carry on with the excision there and then should it be considered feasible. In one or two cases I have made a limited exploration at the time of making the gastrostomy, and as a result have concluded that at a second sitting it might be possible to remove the growth. When two or three weeks later such an attempt was made, the growth was found to have become much more active and was fixed beyond hope of successful removal; it was as if the condition had been stimulated to activity by the previous interference. There is also the question as to whether such growths might be brought within the range of operability by previous radium or x-ray treatment. In the following case the growth was actually removed by this "pull through" plan. The patient was a man of 62, a cheerful fellow and of strong determination and willing and anxious that an attempt should be made to restore his capacity to swallow. The diagrams show better than words what was actually done.

1st step. Fig. 11.

Abdominal exploration. This proved the growth within reach of the finger and its separation from surrounding parts could almost be completed, but it would not pull down and seemed to be firmly attached at some point which could not be reached.

2nd step. Fig. 12.

The abdominal incision was covered with hot towels and attention turned to the neck. The oesophagus was exposed by an oblique incision cutting completely across the sterno-mastoid. This gave a much easier exposure than the classical incision along the anterior border of that muscle. (Fig. 12a.) The cellular plane around the oesophagus was easily identified and the finger introduced, when it was found that the upper limit of the growth could be reached by the finger and separated from the surrounding parts; the oesophagus was cut across about the middle of its cervical portion and the upper end was fixed to the skin margins of the cervical incision. Traction was then made on the lower end to determine whether or not it could be drawn up into the neck, but it did not yield

readily and it was apparent that the final separation and removal must be conducted from below. The upper end of the thoracic oesophagus was surrounded by gauze which was firmly tied

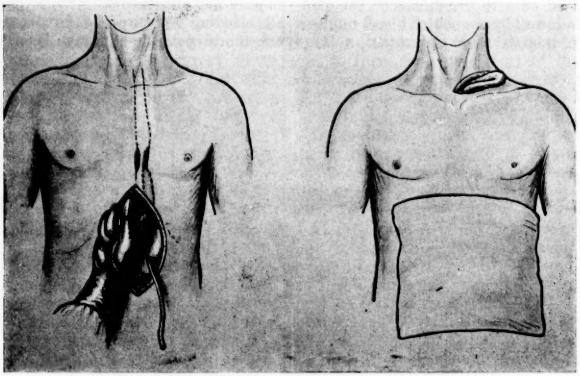

FIGURE 11. The earlier stages of removal of the oesophagus by complete enucleation.

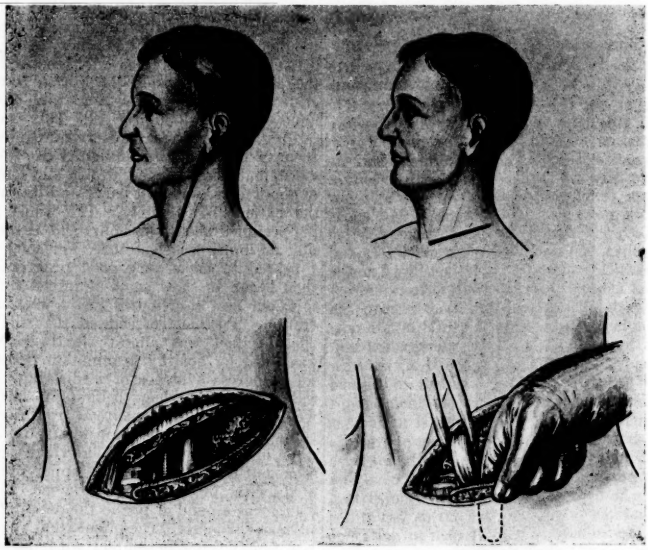

FIGURE 12. The cervical step in the operation for complete removal of the oesophagus. The figure at the upper left is a, that at the upper right is b.

over it, and was then pushed into the mediastinum and the wound above it lightly packed with iodoform gauze and the neck incision largely closed.

3rd step. Fig. 13.

A return was made to the abdominal field with the expectation that it would now be easy to withdraw the separated oesophagus into the abdomen, but it was still firmly attached somewhere, and even firm tugging failed to dislodge it. Finally it was necessary to introduce the whole hand into the posterior mediastinum and

to practice the manoeuvres of the obstetrician separating a retained placenta before it was eventually loosened and could be withdrawn into the abdomen where it hung loose out of the wound, attached only to the stomach. Its withdrawal was followed by a gush of blood but this soon stopped, though as a precaution a large pack of gauze was temporarily thrust into the posterior mediastinum. The oesophagus was now cut away from the stomach with an area of the wall of the latter, and the opening so left in the gastric wall was repaired. Attention was now paid to the mediastinum. The removal of the gauze pack disclosed an enormous yawning cavern in and out of which air rushed with a terrifying and disconcerting noise, but fortunately all bleeding appeared to have ceased. It was not found possible to draw together what had been the oesophageal aperture, but it was effectually closed by applying the left lobe of the liver over it like the lid to a box, fixing it there by a stout suture. It now only remained to close the abdominal incision, of course leaving the gastrostomy tube in situ. A drawing of the part removed is shown in Fig. 14. This major proceeding, which my friends remarked reminded them of the mediaeval indignity of "drawing" a criminal, the "hanged, drawn and quartered" of those days, was well borne and did not upset the patient inordinately. The whole proceeding occupied about two hours and at the conclusion the pulse was just 102. For some days he appeared to make very good progress, but he then began to show signs of infection, and despite our best efforts died just a week after operation. Postmortem showed that the oesophageal tunnel was almost rigidly patent and was lined by healthy granulation tissue. There was no gross evidence of mediastinitis. There was some haemorrhage behind the left pleura, which had presumably spread from the mediastinum, but there was no injury to the pericardium or to either pleura. The right sac showed some purulent lymph in the upper part associated with broncho-pneumonia on that side. There was a pond of pus in the depth of the left posterior triangle. The intact right vagus nerve was found lying in contact with the aorta behind the oesophageal tunnel from which it was separated by a layer of fascia. The whole of the growth had been removed and there was no sign of glandular invasion or dissemination.

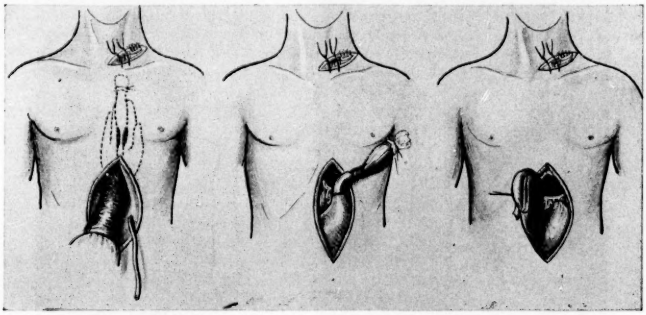

FIGURE 13. The last step in operation of complete removal of oesophagus. The details are given in the text.

FIGURE 14. Malignant oesophagus removed by the pull-through method.

Certain measures for improving or modifying the technique suggest themselves. Whenever possible it would be best to withdraw the

oesophagus from the incision in the neck and to leave the upper end buried in a subcutaneous tunnel to a point below the clavicle. This plan, which was, I believe, suggested by Torek, would very much facilitate the subsequent formation of an ante-thoracic oesophagus and would also prevent soiling of the cellular planes in the neck. The latter appears to me to be a very grave danger, and if the oesophagus must be divided in the neck then I would advocate the following measures to diminish the risks: (1) fixing a tube in the pharyngeal end of the oesophagus to conduct the saliva away from the wound, (2) nursing the patient in a moderate Trendelenburg position, (3) very frequent change of the gauze packing, (4) use of a suction apparatus fixed in the neck wound. Though this patient died, the experience was really a great encouragement. Since carrying out this operation, it has come to my knowledge that E. Gohrbrandt of Germany has completed a similar though not quite identical proceeding in 2 cases, but unfortunately neither of them survived. Nevertheless in reviewing the various other methods he concludes that this "pull through" plan probably holds out the best prospects of success in this admittedly difficult problem. Of course the story is but half told, and to rid the patient of his swallow and to leave him with two fistulae would be like the Negro who in the boom days invested in a dozen sixteen-dollar silk shirts but said "he didn't wear no collars". To add the collar is of the essence of the contract, and I had in mind the various methods of restoring the oesophagus with which I was familiar. I first saw an attempt to improvise an oesophagus made by Caesar Roux in Lausanne in 1908. He used Tavel's method, bringing an isolated loop of jejunum up under the skin of the chest wall as illustrated in Kocher's Operative Surgery. The operation was beautifully carried out, but the patient did not survive and so it has been in the hands of most surgeons, though one or two successful cases have been recorded. In Copenhagen, in 1923, I saw a young woman for whom that great Viking, Professor Rovsing, had constructed a new oesophagus from the skin over the front of the chest wall. In that case the oesophagus had been obliterated as the result of swallowing caustic lye. It was a brilliant success and the patient was fat and well nourished and demonstrated the functional capacity of her new swallow by drinking from a tumbler easily and freely. A similar case was recorded by Professor Zaaijer of Leyden, and in the English *Lancet* for May 4, 1929, he published an illustration of the man eleven years after the operation. One of my countrymen, Mr. H. H. Sampson of Birmingham, England, has also had a similar success and his patient is leading a normal working life and is employed in the hospital laundry. This case was especially worthy of note and is succinctly described in an account of a meeting at Birmingham on page 1291 of the London *Lancet* for June 14, 1930. The girl was suffering from a severe stricture, the result of swallowing caustic soda. The case had been treated with bougies and in consequence a perforation into the bronchus had occurred. The cervical oesophagus was transplanted under the skin to form the upper part of the new gullet while a loop of jejunum was used for the lower part, the intervening segment being formed from the skin of the chest wall. This plan appears to be the method of choice, though of course it must be modified according to the circumstances of each case. These cases show that the pioneer work of doubtless many surgeons has placed this part of the problem on a secure footing.

At this stage it is not possible to define what may be considered the indications for an attempt to excise the malignant oesophagus by this type of operation. In this matter the surgeon must be guided by his own view of the matter. I would only say that in pioneer work, progress is usually made by doing too much rather than too little and profiting by our mistakes while always remembering that

"Knowledge when wisdom is too weak to guide her
Is like a headstrong horse that throws her rider."
(16th Century Complet.)

Was it not Douglas Jerrold who said that "a prudent man is like a pin for his head prevents him going too far?"

But in dealing with cancer we must never overlook the claims of the incurable or abandoned cases, and as a palliative measure I am an enthusiastic advocate of gastrostomy, and I cannot understand the disfavour with which this valuable operation is viewed by so many surgeons. Other methods of palliation such as dilatation or intubation both have their advocates, but as I have no special personal experience of either I will make no comments. However, gastrostomy, after the method of Kader and Senn, has proved most satisfactory, and it has many advantages. Once done no intrabuccal manipulation is necessary; the patient need not look forward with dread to the repeated passage of bougies or to the adjustment of tubes or to the replacement of those that are lost, and all fear of starvation is removed. On surgical principles it appears to me to be far from ideal to manipulate or traumatise a malignant growth in any way, and save for the occasional swallowing of saliva gastrostomy does provide a method of giving absolute rest to the growth. I have carried out the operation in nearly two hundred cases and have been witness to the great benefits conferred and to the absence of unpleasant complications. The attitude to palliative measures seems largely a question of temperament and as much the temperament of the surgeon as of the patient. If those in charge approach the case with an obsession that gastrostomy is an unsatisfactory or

undesirable proceeding, this attitude will react detrimentally on the patient. We may admit that gastrostomy is reminiscent of the small boy's definition of a lie "an abomination unto the Lord but a very present help in time of trouble" but the same holds good of all similar proceedings such as colostomy. I cannot help feeling that many surgeons are developmentally unsuited to deal with inoperable malignant cases, and that their offices might as well be labelled "Abandon hope all ye who enter here." The proper attitude is more nearly defined by your physician poet when in "The Morning Visit" he bids us

> ". . . last, not least, in each perplexing case
> Learn the sweet magic of a cheerful face."

The surgeon should remember that if the patient has an urgent desire for food, gastrostomy is always worth while. Entire absence of appetite probably portends secondary deposits and means that the beating of the wings of the Angel of Death will soon be heard approaching. I remember with shame the case of an old man, in his eighties, whom I saw in a small room high up in a city dwelling. He was suffering from oesophageal obstruction which had rapidly become absolute and had brought him to a state of starvation in which he was clamouring for food. Because of his age and condition I fell into the trap of agreeing that an operation would not be worth while, and to this day I remember with a shudder that it was several days before the pangs of hunger were relieved for him by a merciful death. In cases of this kind there is no excuse for withholding gastrostomy, and as William Mayo said years ago, in cases which are too ill to investigate there is always the chance that the diagnosis may be wrong! I remember more than one instance of this sort, and especially a man of 56 who was admitted to hospital in 1894 with a history of difficulty in swallowing of 4 months' duration, followed by great loss of weight and exhaustion. The diagnostic methods of those days left probability full sway and the diagnosis of malignant disease seemed almost certain. Gastrostomy saved his life and he was soon lost sight of, but more than seven years later he unexpectedly turned up quite well and strong and able to do his work as a horse van driver. He stated that some months after leaving hospital he turned tired of using the gastrostomy, and as he found he could eat enough soft food to satisfy his appetite, he removed the tube and allowed the fistula to close.

When I commenced to explore cases of malignant oesophagus, I had hopes that it might sometimes be possible to short-circuit the growth by making an anastomosis between the dilated oesophagus above and the fundus of the stomach below. So far this has not proved possible, but I am not without hope and I think that perhaps induced paresis of the diaphragm might facilitate this proceeding.

Many years ago that man of great foresight, John Bland-Sutton, opened up a fascinating vista by his reference to cases illustrating the importance of "Environment in Pathology" and there is no more apt illustration of his theme than the conditions affecting the oesophagus. Progress in this field teaches us ever to bear in mind that the oesophagus is subject to the laws of surgical principles and that its peculiar problems are not inherent in its diseases, but are the price of its environment.

Eventually oesophageal surgery will come into its own, but in connection with its development we must remember John Hunter's dictum, "Why think; why not try the experiment." Development will be slow, for it is an attack on one of the last strongholds, and much that has been done and will be done, must doubtless be superseded. We must be prepared for this sequence and its contemplation reminds me of that word monument of your Oliver Wendell Holmes where he so beautifully expresses the idea of the painful and rejected steps on the upward trend to perfection in his "Chambered Nautilus."

> "Year after year beheld the silent toil
> That spread his lustrous coil:
> Still as the spiral grew,
> He left the past year's dwelling for the new.
> Stole with soft step its shining archway through,
> Built up its idle door.
> Stretched in his last found home,
> And knew the old no more."

But let us avoid the error of neglecting to honour the steps by which we rise. These personal experiences in this branch of surgery are offered with great diffidence. The circumstances under which I work debar me from the opportunity of concentrating attention on any one problem and I am well aware of the incompleteness of my efforts here recorded. Such as they are I offer them as an incentive to others and in the hope that they may stimulate interest and enterprise. It appears to me that you in America with your well-equipped clinics, ample material resources and alert minds ought to play a principal part in the development of this fascinating branch of surgery.

MASSACHUSETTS MEDICO-LEGAL SOCIETY

SUDDEN DEATH IN A CASE OF ASYMPTOMATIC SUBACUTE BACTERIAL ENDOCARDITIS*

BY GEORGE LEAN WEST, M.D.†

A WOMAN, 31 years of age, while playing tennis with her husband suddenly stopped the game, walked in a natural manner to the side of the court and sat down. She fumbled at her shoe-lacing and then called to her husband, "Come over here." To her husband's question, "What is the matter?" she replied, "I don't know; I feel sick." With these words her body relaxed, and she sank slowly to the left side, became unconscious and could not be aroused. She immediately became restless, making vague indefinite movements of both arms and legs, not of a convulsive nature. The physician who was summoned arrived quickly and found her cyanotic, pulseless, but still breathing. She was taken without delay to a hospital within approximately ten minutes' ride, but was dead on arrival.

Investigation disclosed the facts that the woman was an athletic instructor, that she was an expert swimmer and had lately been sought as a swimming instructor for a large educational institution. She played tennis and rode horseback regularly. Her husband never knew of his wife's being ill or making any physical complaint. She had consulted a physician three years before her death for a wrench of the neck muscles due to an automobile accident. While in Honduras several years previously, she was thrown from a horse and she was supposed to have had malaria in Porto Rico.

On the day of her death, she was in her usual health, had eaten a hearty dinner and ridden in an automobile about five miles to reach the tennis court. The game had just begun as she showed her first sign of distress.

The autopsy showed a female 5 ft. 6 in. tall with an estimated weight of 120 pounds. She was of athletic build and her narrowed hips gave a suggestion of masculinity.

The pupils were equal, regular and 6 mm. in diameter.

The stomach contained approximately 330 c.c. of partially digested food and the mucous membrane was normal in appearance.

The liver, gall bladder, pancreas, spleen, adrenals, ureters, bladder, uterus and adnexa, and the intestines were grossly negative.

The pleural cavities each contained 5 c.c. of clear, straw-colored fluid.

The right lung weighed 620 gr. and the left 600 gr. Both lungs were distended, non-crepitant and deep red in color. The cut surfaces yielded a considerable amount of frothy sero-sanguineous fluid on pressure.

The thymus was present and weighed 10 gr. The thyroid was not enlarged.

The brain weighed 1300 gr. The meninges appeared glistening and moist. No lesion of the brain substance was found.

The heart weighed 340 gr. The epicardium and the coronary arteries showed no gross pathology. The myocardium appeared normal. The sizes of the valves were within normal limits and the endocardium was normal except in the region of the aortic valve. On the under surface of all the aortic cusps were friable vegetations. The anterior cusp showed a vegetative growth 2 mm. in length, 2 mm. wide and 5 mm. thick, pinkish red in color and easily detachable. A single vegetation 3.5 cm. long, 1 cm. wide and 1 cm. thick, occupied the under surface of the other two aortic cusps. This mass had ulcerated and had left a deep ulcer with irregular roughened walls. At the bifurcation of the left coronary artery was found the cause of death, an embolus of vegetative growth from the aortic valve, which was triangular in shape, measuring 10 mm. x 5 mm. x 5 mm. and rested in the bifurcation in such a way as to occlude both branches distal to the bifurcation.

Microscopical examination of the liver showed congestion of the sinusoids, and sections of the spleen congestion of the pulp. The kidney sections showed healed glomerular embolization and the lungs showed edema. The vegetative growth, both on the aortic valves and in the embolus showed endocarditis with few widely scattered Gram-positive cocci growing singly and in chains, but attempts to obtain cultures from the blood or from the vegetations met with no success. The fatal lesion was subacute-bacterial endocarditis with vegetative embolus to the left coronary artery.

Embolism is a prominent happening in subacute bacterial endocarditis, the smaller emboli appearing as petechiae in the skin, more particularly at the base of the neck and along the edges of the fingers or toes, or in the conjunctivae. Larger embolic masses are capable of giving abdominal pain and localized peritonitis when they infarct the spleen. Glomerulonephritis is common, and even abscesses of the kidney may arise from infarcts in that organ. Infarction as a result of emboli from the vegetative growth may cause blindness, aphasia, or lung abscess. Embolic occlusion of the coronary artery in the course of the disease as a cause of sudden death is rare.

These various embolic manifestations are associated generally with prostration, which begins insidiously with weakness and malaise and later is accompanied by fever, generally prolonged and of an intermittent type, conjoined with chills and sweating. The kidney becomes the seat of glomerulonephritis. The heart responds to the invasion of the valve cusps and chordae tendineae, by murmurs, which are various in intensity and character. The blood shows a culture of the streptococcus viridans in 95% of the cases. Secondary anaemia sets in, the patient becomes profoundly septic, and death occurs in a few weeks or months.

It is interesting to note how wide is the variance between the ordinary course of the disease and the course of the disease in the case under consideration. Instead of weakness and prostration, we find here strong, vigorous, abounding health and vigor in a young woman who was

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eager for sport and entered heartily into her game. To her husband's knowledge she had never consulted a physician except in an instance of personal trauma. While it is fair to assume that an aortic valve so seriously affected must have given evidence by some murmur, no physician had had the opportunity to hear that murmur and to investigate its cause. It is equally true that, despite the deformity of the valve and a very probable murmur, the circulatory disturbance arising from the valve affection was not enough to give any warning. The fact of the apparently undisturbed tenor of her active life, the fact of the presence of the vegetations on the aortic valve and not the mitral valve, the fact that the wall of the left ventricle measured only 5 mm., that the heart cavities were of normal size, that the heart weighed 340 gr., are evidences that unrecognized rheumatic conditions of childhood years on which the present bacterial conditions might have been implanted, could not have been present. She was never known to have made any complaint of physical ailments other than very minor ones. She pursued her occupation as athletic instructor. The presence of the healed embolic manifestations in her kidneys, although few in number, showed that at some time the blood stream must have contained some infective material, presumably from the vegetative growth on the aortic valve. The emboli were not, however, in sufficient numbers to disturb the normal efficiency of the kidney. It appears here as a most interesting observation that the first symptom by the subject of the disease was the distress which she showed while playing tennis and the distress passed quickly into the objective signs of coma, cyanosis, and death.

With the first conception of subacute bacterial endocarditis as a distinct disease entity, following the studies of Schottmuller[1], and later by Osler[2] and Libman[3], came the idea of absolutely fatal prognosis. Later studies by Libman[4, 5] and Weiss[6] have led to a marked change in the prognostic conception. In many cases the pathology in the heart has shown thickened, shortened and retracted valves covered with various vegetative growths of firm, granular, friable masses in an organized form, with deep ulcerated areas filled with organized or even calcified material, and yet the subjects of these conditions have survived and led active lives and have finally succumbed to death from causes not assigned to subacute bacterial endocarditis. It is a well-known fact that in some cases the culture of the blood may show the streptococcus viridans for some time and later, without medication, the blood may become bacteria-free. Libman has reported the recovery of several patients who have had fever, enlarged spleen, petechiae, tender nodes, embolism, and progressive anaemia. Soma Weiss has reported the presence of the classical picture of subacute bacterial endocarditis in two cases of young men in apparent good health who had come to their death from violence.

It would appear that our present conception of subacute bacterial endocarditis must be that it is a disease with manifestations of varying severity, ranging from fatal cases with exaggerated symptomatology, to severe cases with marked symptoms, in which through increased resistance, recovery may take place; and finally, to those cases where there is a complete absence of subjective symptoms and signs. The case under consideration is interesting in that it presented classical pathology in the endocardium but no symptomatology until a few minutes before death, when embolism of the coronary artery occurred as a direct result of the ulceration and detachment of the vegetative growth.

From the studies of this disease it would seem probable that the number of silent cases is by no means small, and that many go on to recovery. It certainly is rare to have a case terminate as did this one. Yet, having once occurred, it is of tragic interest to know that a young person in vigorous health may still carry within his own organism a lethal weapon which, with startling rapidity, may accomplish his death.

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Discussion

Dr. Leary: This case is interesting from several standpoints. First, it is a case of coronary obstruction. I recently checked up 100 cases of coronary deaths, which indicated that thrombosis over an atheromatous focus with edema which had almost closed the lumen was the commonest cause of sudden coronary death, as was to be expected. In a large series, however, coronary inadequacy due to marked narrowing of the lumen owing to arteriosclerotic changes was the important factor. In this group unusual stress played an important part in causing the death. The circulation was adequate under ordinary conditions, but when unusual conditions supervened was no longer sufficient. For example, a woman was found dead in a closed apartment room where a gas stove was burning with all burners full on. With her were two men. All had been drunk. The carbon monoxide which accumulated did not seriously affect the men, but the woman with a coronary sclerosis and inadequacy died from the

combination of carbon monoxide poisoning, alcoholism and the coronary fault.

My series included three cases in which an aortic vegetation plugged a coronary orifice and led to the death. Obliteration of coronary orifices, partial or complete, due to syphilitic aortitis is a not infrequent cause of sudden death, although it is amazing how the circulation can be cared for through the thebesian veins in this condition, since the encroachment on the orifices is slow.

Coronary embolism is rare, this series including only a single case in which a small molded embolus, evidently arising from an auricular lacuna, was responsible for the obstruction.

This case is also interesting from the standpoint of subacute bacterial endocarditis. This condition arises, as indicated by Dr. Levine of the Brigham Hospital staff, in individuals who have suffered from rheumatic endocarditis and who have overcome the rheumatic process; who, in other words, have become immune to rheumatism. The condition is remarkable in that its victims can walk about and carry on work for months or even for some years after infection. A girl comes into the laboratory for a blood culture, who, while somewhat pale, does not appear to be sick. Among a group of girls she would not be picked out as ill. However, 1 c.c. of blood on an agar plate produces innumerable colonies of streptococcus viridans.

The organisms isolated from these cases are not virulent enough to produce infection in experimental animals which are susceptible to infection with other strains of streptococcus viridans. The condition seems to be a symbiosis between the patient and the bacterium. Kinsella has shown that the living organism, when injected subcutaneously into the patient himself, provokes no more reaction than would an injection of saline solution. The body as a whole is immune, but the endocardium is susceptible. Death is due to accidental causes. A mitral vegetation becomes so large as to mechanically obstruct the valve orifice, or death may be due to kidney insufficiency arising as the result of myriad glomerular infarcts. The glomeruli do not react to the emboli producing these infarcts as tissues should react to bacterial emboli. The tissue response is that which should arise from bland embolism. An unusual accident producing death is coronary obstruction by a vegetation, as occurred in this case. Even among cases of subacute bacterial endocarditis Dr. West's case is unusual, since the woman was an athletic instructor and had not, apparently, been ill in any sense.

DR. MAGRATH: My own experience with coronary embolism just about matches Dr. Leary's. I recall an instance of aortic mural thrombosis, of low grade, which had for its locus a spot so near the left coronary that it had slipped over the opening and closed it; there was no actual embolism. Coronary disease is a prominent factor in sudden death; it is of marked practical significance in connection with life insurance. For example: A man, 33 years of age, an underwriter, who up to the last day of the year carried $25,000 life insurance, in February passed a medical examination for another $25,000. A few weeks later he came from his office to the Parker House to attend a meeting and at once complained of feeling as if he had a little indigestion. He took something for it and sat down near a window, asking someone to open it. The cigarette which he was smoking dropped from his hand and he was dead within twenty minutes. I learned afterward that he had been to a well-known clinic during the summer and had had a rather thorough examination. This included a note of coronary abnormality as indicated by electrocardiography. The Wassermann test was negative. He died from endoarteritis of the luetic type. This was a man who had successfully passed an examination for life insurance within six months of his death.

In another instance a woman, young and supposedly in the pink of condition, walked from her office down town to lunch with a friend. She complained of a little substernal pain and said she hoped she would not have pneumonia. She spent a very pleasant afternoon and evening with friends and joined her roommate at their apartment and talked with her until 1:30 in the morning. The roommate left at 8:30 and thought that she was sleeping. Later on it was found that she had died in her sleep. Postmortem examination disclosed extensive aortic and coronary disease (luetic in type).

DR. LEARY: The importance of what is thought to be indigestion in cases of coronary sclerosis cannot be overstressed. Symptoms of gastric pain and distress are more common than the typical syndrome of angina. Attacks of acute indigestion without organic disease of the abdominal organs in men over 50 should lead to immediate suspicion of coronary disease. I have said before that the Seidlitz powder is a most deadly weapon in cases of cardiac distress.

(Demonstration of heart showing coronary sclerosis and inadequacy from the case of carbon monoxide and alcohol poisoning mentioned.)

SPEAKER FROM THE FLOOR: I can cite an instance of a gentleman who died under similar circumstances, a successful shoe salesman who had passed life insurance several weeks before.

DR. DOW: A patient of mine, who took his Ph.D. at Harvard, went to New York and dropped dead thirty days after receiving his degree. His death certificate came back with the notation "coronary obstruction". Thirty days before he graduated one of the companies wrote out life insurance on him. Previous to that he was put out of the navy because of some "heart trouble".

(Specimens shown by Dr. Magrath.)

DR. MAGRATH: Reference to traumatism in relation to these cases reminds me of the case of a man alleged to have been hurt in an automobile accident, with some injury to his head, following which he had attacks diagnosed as petit mal. The day before yesterday he suddenly collapsed and died on the accident room table. The autopsy was negative except for a very narrow, tortuous coronary artery. There was no lesion of the brain.

DR. LEARY: Fainting attacks do occur in coronary disease. I remember the case of a veteran who underwent repeated anginal attacks followed by fainting. He was looked upon by the Veteran's Bureau as a malingerer. He died, however, in one of the attacks, and malingerers are careful not to die. Postmortem examination disclosed no adequate cause of death. The coronary arteries were normal. I look upon this as one of the rare cases of coronary spasm, giving rise to typical angina and leading to death during an anginal seizure.

DR. MAGRATH: A few years ago this happened down on Sudbury Street in a so-called laboratory where perfumes were manufactured. Alcohol was stored in the rear of the laboratory and a fire broke out. The manager, thinking of the alcohol, went back to warn the firemen but did not stay long. As he came down the stairs, he said, "I am dying, get me to a hospital." He was taken to the Haymarket Relief Station where, on examination, he appeared to be suffering from angina pectoris. As he was being put to bed and while assisting the orderly in the process of undressing, he suddenly fell backward onto the bed, dead. Autopsy disclosed no organic disease and no changes either in the heart or in the coronary arteries explanatory of what was undoubtedly a "cardiac" death—death from *asystole*. If there is any such thing as *spasm* of the coronary arteries, I am certain that this is an instance of it.

SUMMARY OF THE MIDDLE-RATE PLAN FOR HOSPITAL PATIENTS — THE FIRST YEAR'S EXPERIENCE OF THE BAKER MEMORIAL OF THE MASSACHUSETTS GENERAL HOSPITAL, BOSTON, MASSACHUSETTS

BY C. RUFUS ROREM

How the Massachusetts General Hospital in Boston has made it possible for families of moderate means to pay their hospital and doctors' bills for serious illness is the subject of a report by C. Rufus Rorem, published by the Julius Rosenwald Fund, which is coöperating in the experiment.

The aim of the middle-rate plan reported by this study is to minimize the total costs of hospitalized illness, including the fees for physician and special nurse, by consolidating costs, furnishing service on a sufficiently large scale to permit economy in administration, and offering it without profit and without charity.

"People who are self-supporting during health and who may be supposed to have at least a normal pride in independence will presumably prefer not to be recipients of charity when some member of their family is sick," declares Dr. Michael M. Davis, director for medical services of the Julius Rosenwald Fund, in a preface to the report. "Amid the extensive discussion among the trustees, physicians, and superintendents of hospitals as to how to provide for the much advertised 'person of moderate means' an attempt to raise money to meet his needs has been more prominent than the effort to devise a service the cost of which he could pay for. The middle-rate plan was inaugurated to accomplish the latter objective—namely, to provide services to hospital patients at moderate rates rather than to help them pay for their care. . . . For the nation at large the method of endowment is no more practical on the financial side than it is in accord with the American principle of self-support."

According to this plan, inaugurated in March 1930, the Massachusetts General Hospital provides private and semi-private rooms for middle-rate patients in its Baker Memorial Pavilion. During the first year's operation nearly 2,500 patients were treated, representing the lower-salaried professional groups, "white-collar" members of business groups, artisans, elderly persons living on small incomes, and the like. The average family income was $2,500. In accepting patients, consideration is given by the hospital's admitting officer to their family income, savings, debts and obligations, expenses for past illness, the probable cost of the present illness, the probability of the patient's complete recovery or possibility of his future dependence, and the probable length of time before he will again be able to earn money, if his income stopped because of his illness.

A distinctive feature of the middle-rate plan is that the patient receives only one bill, which includes the doctor's and all other charges. Room charges, including usual medicines and regular nursing service, are from $4 to $6.50 a day. Physicians and surgeons on the staff of the Massachusetts General Hospital have established a moderate schedule of fees for patients treated in the Baker Memorial Pavilion and have further agreed that the maximum professional charge to any one patient is $150, no matter what the length or complexity of his illness. Special nursing, required only in exceptional cases, is furnished at the rate of $6 a day for the nurse and $1.50 for her room and board. Flat rates cover the usual laboratory and other special fees, while unusual services or drugs are provided at cost price. All items are included in the bill rendered to the patient by the hospital, with provision for instalment payment if necessary. The hospital collects the whole bill and turns over

(Continued on page 688)

PREGNANCY AND FIBROMYOMA

Report of a Case Complicated by Rectal Obstruction

BY SAMUEL R. MEAKER, M.D.*

FIBROID tumors of the uterus are commonly associated with impaired reproductive function. The coincidence of these neoplasms and sterility is recognized, although one cannot define a causative relationship between the two conditions. When conception occurs in the fibromyomatous uterus, special complications may arise during pregnancy, labor, or the puerperium. Nature is often extraordinarily adroit in her management of such cases; nevertheless, the aid of the obstetrician sometimes becomes necessary.

The following case is reported because of the number of its unusual features.

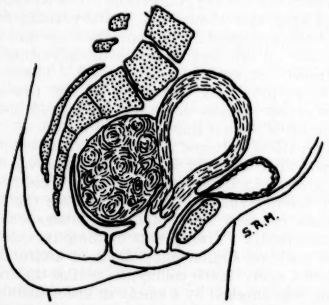

FIGURE 1. Sketch showing anatomic relationships of tumor. It is incarcerated under the sacral promontory, and is compressing the upper two-thirds of the rectum.

REPORT OF CASE

A nulliparous married woman of 42 was seen in consultation with Dr. John S. Martin on January 12, 1931.

Her past history was uneventful. The present illness centered around two complaints, amenorrhoea and constipation. Menstruation had been regular and normal up to and including the period of October 5, 1930, since which there had been no flow. The breasts became enlarged and tender, and in December some morning nausea appeared. Vesical symptoms were absent. Constipation began to be troublesome around the middle of December, and increased in severity until ribbon-stools were passed and movements were obtained only with the greatest difficulty. The stools contained occasional streaks of blood.

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Examination showed the uterus to be in first-degree descent. Its size, shape, and consistency corresponded to a pregnancy of about fourteen weeks. Behind the uterus lay an irregular hard mass nine centimeters in diameter. This appeared to fill the hollow of the sacrum, and was practically immovable. X-ray after barium enema demonstrated a marked rectal blockade, but gave no suggestion of an intrinsic neoplasm of the bowel.

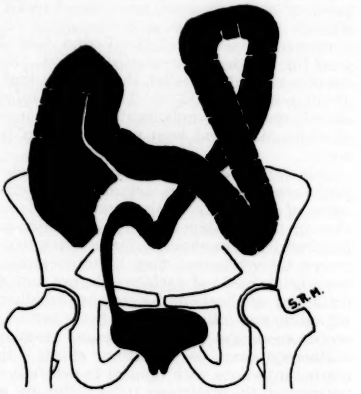

FIGURE 2. Skiagram of barium enema. The upper rectum is compressed, and is displaced to the right. The transverse colon and caecum are "domed" over the enlarged uterine fundus.

These findings were the basis for a diagnosis of fibromyoma of the pregnant uterus, incarcerated under the sacral promontory and obstructing the rectum. Interference was clearly indicated, and accordingly operation was undertaken on January 22. The tumor proved to be attached by a broad pedicle low on the posterior face of the uterus. No special difficulty was encountered in the performance of myomectomy.

Happily the pregnancy continued without disturbance. On June 30 the patient was delivered by caesarean section, of a normal female child.

SUMMARY

This case presents the following unusual combination of circumstances: elderly primigravidity; pregnancy and fibromyoma; incarceration of the tumor; rectal obstruction; successful myomectomy at four months; and ultimate delivery at full term.

LABORATORY STUDY OF THE GONOCOCCUS*

Clinical and Bacteriological Study Made of a Special Group of Cases†

BY BENEDICT F. BOLAND, M.D.,‡ AND WILLIAMS COCHRAN, M.D.‡

GONORRHEA, which causes one-third of the pelvic invalidism in the female, still continues to be as prevalent as ever. Treatment of this condition has not varied markedly and hot douches and local applications of different types constitute the principal armamentarium for its eradication. For the purpose of study, a group of over fifty cases were placed under the writer's supervision in a special clinic of the gynecological service of the Boston City Hospital for over a fifteen months' period. No attempt was made to select the cases so that the group was composed of the freshly infected, chronic and pelvic inflammatory types, in some of which there had been some depletion treatment.

This investigation was instituted as an unprejudiced attempt to determine the clinical value of diathermy in the treatment of gonorrhea in the female. So that the results might be carefully cross-checked, the investigation embraced three phases: first, bacteriological, cultures being made of each case before and after diathermy application, to determine whether the organism was killed by local heat; second, observation on the patient's clinical reaction to diathermy and its resulting effects; third, uterine injections with lipiodol to determine the patency of the Fallopian tubes following a so-called cure.

This paper is confined to the bacteriological phase in an endeavor to learn the advantages of cultures over smear examination, the viability of the organism, and to experiment with media and cultures as a laboratory adjunct in the treatment of this disease.

These cases were cultured and the gonococcus isolated and regrown on similar media for at least 48 to 72 hours. The cultures were then placed in suspension and an agglutination test employed to be sure that the organism was really the gonococcus. Smears were made of both the original culture and the transplant and stained by the gram method; the typical gram-negative diplococcus was found. Nearly 1000 cultures were studied to rule out other organisms, namely micrococcus catarrhalis and meningococcus, both of which resemble the gonococcus in smears.

Modern advances in the various media used in the laboratory have enabled the bacteriologist to grow the gonococcus in culture as readily as any other organism. Bumm, in 1885, was the first to cultivate this organism in pure culture and did so upon coagulated human blood serum. After cultivating several generations, he proved its virulence by inoculation in man. Moisture, proper type of agar, and the correct temperature are essential requisites.

Hydrocele or ascitic fluid agar, blood agar, brain veal and North gelatine agar, each proved satisfactory as a medium for this organism. Each of these media had sufficient moisture, which was essential for the gonococcus. The North gelatine agar and brain veal in dehydrated form are both produced by a manufacturer and can be made up into agar slants after adjusting the hydrogen ion concentration to 7.8. These two latter preparations, however, did not prove so satisfactory as the ordinary laboratory media made up of ascitic fluid or blood agar.

About 200 cultures made of the above media were studied in order to acquaint ourselves with the growth in these various agars. This procedure readily enabled one to segregate the gonococcus colonies from other organisms on the culture media. Transplants of the pure colonies were made on similar agar slants of ascitic fluid or blood agar. Each colony, as well as the transplant, was checked by a smear in order definitely to prove the organism to be a gonococcus culture.

Original pure transplants were tested for viability and daily cultivation was made to determine the length of time this organism would grow. Pelouze[2] contends that the germ will survive from three to six weeks on these sub-cultures, but our experience proved four days to be the maximum period. Results obtained on blood and ascitic agar were compatible with brain veal agar, which Pelouze maintained was the best medium. This procedure was tested in various batches of media and four days still remained the maximum period regardless of the media used. These checks were made to rule out the possibility of error in technique in preparation of the various batches of media. Ordinary cornstarch in small percentage, or powdered charcoal added to agar, made a suitable medium for growth, but neither was so perfect as the ascitic fluid or blood agars.

Cultures on these various media, especially the brain veal and ascitic fluid agars, were transplanted in pure culture and subjected to heat in a water bath, for periods of one-half an hour. Series of transplants of the same cultures were used and each series was subjected to a constant

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temperature ranging from 40° C to 45° C. All five series were replanted after the heat experiment on other slants of the same medium and growth was produced by the ordinary incubation procedure upon the new slant. The fact that these cultures still survived after heating at such a high temperature was also confirmed by the fact that cultures taken four weeks later from the same patient after four treatments with diathermy produced colonies on the same media. The conclusion, therefore, was that heat alone at high temperature did not kill this organism.

A similar set of cultures from the same series was placed in the ice box for a period of five to ten hours, submerged totally in ice. These transplants also grew when subjected to the usual laboratory methods of incubation. Low temperature also failed to have any effect.

Smears—Each patient, after having a clinical and smear diagnosis of Neisserian infection made, was transferred to this series. Sterile swabs were used to take the culture and the plant was made on a square in a Petri dish, which had previously been divided into four squares. A culture from both the cervix and urethra was taken before and after treatment with diathermy. The swab was also used to make a smear for clinical examination. Cultures, after incubation, were examined for colony growth and a finely granular, grayish-white and sometimes yellow-tinged colony picked and a smear made for gram staining. If the smear proved to be a gram-negative diplococcus, the colony was transplanted to make a pure culture. If the smear was positive, the pure culture exhibited typical gonorrheal cultural growth. By this method when the clinic technician's smears were positive and, in turn, corroborated by cultural growth. Smears and cultures, therefore, invariably coincided and as much reliability could be placed on a carefully stained smear, if done by the proper technique, as on a culture.

One essential factor in cultural procedure proved to be that moisture of the medium was required, and the culture medium must be made fresh weekly to sustain its moisture. Media over ten days old failed to produce satisfactory growth for these reasons.

We, therefore, considered the distribution of culture media by local Boards of Health to be of no avail since the media might deteriorate through lack of moisture before being used by the general practitioner and produce no growth. Attempts to plant the organism on media from a swab taken three hours previously in the Clinic failed to produce positive cultures readily. Direct planting of the swab on the medium at the time of taking produced excellent growths and in good amounts, even though the medium was not incubated until three hours later. Attempts to secure positive cultures from a swab planted twenty-four hours later, after being carried in one's vest pocket for that period, also failed.

Agglutinations were carried out with a series of eight tubes, the first being a 1/100 dilution and ranging up to the seventh tube, which was a 1/6400 dilution. The eighth tube was a control tube of the organism and saline and compared with these other tubes after thoroughly shaking for five to ten minutes and incubation at 55° C over-night. The average agglutination ranged between a four-hundredth and eight-hundredth dilution.

Further evidence gained by this laboratory procedure showed the means of checking a positive gonorrheal smear by cultural growth, sugar and agglutination tests, as a means of arriving at a conclusion. This check offered definite proof especially in relation to court procedure in divorce cases—where the time-honored phrase "morphologically resembling gram-negative diplococci", is not only indefinite but vague in the last analysis. Laboratory tests plus smear examinations will afford positive proof in this type of court case.

Three cases of endocarditis due to this organism—one of which was an original clinic case—were checked by these methods after the gonococcus was found in a blood culture.

Two of these three patients died, one being autopsied and the case proved by laboratory methods, while on the second patient autopsy was refused because the husband was an undertaker. The third patient recovered and was discharged to the out-patient department, where follow-up work is now in progress on her cardiac and joint conditions.

CONCLUSIONS

1. Modern laboratory methods in well-trained hands can readily grow the gonococcus in culture.
2. Hydrocele fluid or blood agar proved equally good for cultural growth.
3. Heat of 40 to 45 degrees centigrade did not kill this organism.
4. Ice refrigeration for ten hours was also not effective in prohibition of growth.
5. Smears, carefully done, proved as reliable as cultures.
6. Moist media and media made fresh each week were essentials for satisfactory growths.
7. Agglutination proved highly effective as a check on cultures and smears.

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THE NEW HAMPSHIRE MEDICAL SOCIETY

TREATMENT OF PERITONITIS RESULTING FROM PERFORATED VISCUS*

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ACUTE suppurative peritonitis results from an inflammation caused by infection with pyogenic organisms. The organisms usually causing peritonitis are those habitually present in the intestinal tract, the colon bacillus, streptococcus pyogenes and staphylococcus pyogenes aureus being responsible for the majority of such infections. Not infrequently cultures will show a combination of more than one of these organisms in an individual case. Primary infection of the peritoneum is uncommon, as infection usually occurs secondary to injury or disease of some abdominal viscera.

This paper will consider only infections resulting from perforation of a hollow viscus, and no reference will be made to peritonitis resulting from tubal and ovarian infection or from the necrosis of solid organs.

Trauma may cause perforation of any portion of the gastro-intestinal tract, of the gallbladder or of the urinary bladder. In addition to gastric and duodenal ulcers, perforating ulcers may be caused by typhoid fever, tuberculosis, syphilis, foreign bodies within the canal, diverticulitis and carcinoma. Perforation of the appendix may occur suddenly or gradually. When any perforation occurs, the degree of peritonitis resulting from the leakage of foreign matter into the peritoneal cavity will depend upon several factors. If the perforation is very tiny and another peritoneal surface, such as the omentum, is adjacent, an extremely localized peritonitis may occur, a protective zone may be built up around the area, and spontaneous cure follow. This process may not seriously disable the patient.

On the other hand, the perforation of a large gastric ulcer, an enlarged and infected gallbladder, or traumatic rupture of the urinary bladder may suddenly release a flood of irritating and infected foreign fluid into the peritoneal cavity.

Between this last catastrophe and the minor localized peritoneal irritation all degrees of infection may occur. In addition to the factors just enumerated, the extent and degree of the peritonitis also depend upon the type of infective organism and upon the resistance of the patient.

In the development of peritonitis, there is first a phase of peritoneal *irritation* which initiates a serous discharge. This is followed by fibrin and leucocytes. Later the cells begin to die, the bacteria multiply, and the process then becomes suppurative. In localized peritonitis there is developed a surrounding area of adhesive (protective) peritonitis in which fibrin predominates, walling off the lesion from the general peritoneal cavity.

In general peritonitis the phase of irritation is shorter and more active, and extends so rapidly that no adhesive peritonitis occurs. Fluid predominates, first serous, then seropurulent and finally suppurative, and the situation later is further complicated by paralysis of more or less of the intestine, dependent upon the extent of the infection. Paralysis and gas production cause distention. The paralysis is due partly to toxic action on muscle tissue and partly to reflex inhibition as the result of pain. Peritonitis is dangerous directly in proportion to absorption of toxin. It is not the inflammation of the peritoneum that is fatal but the toxins which are absorbed that cause the severe manifestations.

SYMPTOMS

General peritonitis secondary to localized peritonitis may develop after several hours or days, and the onset is gradual. In peritonitis caused by acute perforation the onset of severe symptoms is usually sudden. In many cases these symptoms may be preceded by those of the causative factor. When previous disease symptoms are present they are of great value in determining the initial treatment. Later the onset of diffuse peritonitis will cover up the antecedent group of symptoms.

The diagnosis is made from the history and the usual local and systemic symptoms. In addition to the history it is important to recognize the significance of the rigid retracted abdomen, the type of severe pain and the quality and rate of pulse when the patient is seen early, as this is the period in which lives may be saved by prompt action. The temperature reading is of little importance in diagnosis but a rising pulse means much. The leucocytic count rises, usually to between 10,000 and 20,000 and when suppuration is established the polymorphonuclear count shows 75% or over. Prognosis is more favorable with a white count at or above 20,000, and unfavorable if below 12,000.

X-ray evidence of pneumoperitoneum is positive proof of perforation. Recent x-ray inves-

*Read at the Annual Meeting of the New Hampshire Medical Society at Manchester, May 19, 1931.

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tigations have proved the possibility of locating walled-off abscesses within the abdomen as well as pus in the pelvis and flanks. Fortunately, laboratory aids are rarely necessary in making a diagnosis of peritonitis resulting from perforation.

As suppuration develops and the intestinal muscle becomes atonic, the retracted abdomen is replaced by gradual distention. Pain is an early symptom, and becomes less intense after suppuration has developed. Vomiting may or may not occur early. In the later phases it is common and persistent.

TREATMENT

All patients who develop peritonitis from perforation will die unless operated upon. Whether immediate or somewhat delayed operation should be performed depends upon the condition of the patient. The usual exceptions to immediate operation are (1) severe shock, especially from gallbladder perforation or lower abdominal perforation, (2) signs of localization in an untreated case after 48 hours.

If the patient is in shock from sudden perforation, morphine should be administered, external heat and local heat to the abdomen applied, and intravenous saline solution administered. One to two litres of the solution should be given, the amount depending upon the blood pressure. Intravenous salt solution is always an excellent preliminary when there is even slight shock.

Assuming that we have a patient who has developed diffuse peritonitis from the rupture of a viscus and that the peritonitis has not existed too long to expect help from surgical intervention we must make an estimate of the situation. It is important that much consideration be given the symptoms preceding the onset, as this will determine the site of the incision, and probably the method of dealing with the infection after the abdomen is opened.

Preparation for operation should be as brief as possible. No enema should be given unless for some definite reason. Shaving and preanesthetic medication are all the preparations necessary. If the patient's condition is poor, subcutaneous saline solution should be started at the beginning of the operation and continued throughout. After perforation it is important to keep the patient in a position that will facilitate gravitation of fluid to the pelvis. This precaution is often neglected.

ANESTHESIA

Methods of producing anesthesia are multiplying and today we have a wider choice than ten years ago. Local anesthesia should not be used as it permits very limited intra-abdominal manipulations. From the standpoint of perfect anesthesia, complete relaxation and the absence of postoperative discomfort spinal anesthesia is to be preferred. There are two objections, however, to this method. Spinal anesthesia lowers the blood pressure, and the Trendelenburg position required for this type of anesthesia reverses our ideal posture for gravitating fluid to the pelvis. In early cases, during the stage of irritation, these objections are very real. After diffuse suppuration has developed these objections are less important or cease to exist. The alternative choice of anesthesia is gas-oxygen alone; with ether; or ether alone. My personal choice is spinal anesthesia in spite of the objections mentioned.

If the patient is vomiting or the stomach is dilated, a Levin gastroduodenal tube should be introduced through the nose into the stomach and the stomach emptied before operation. The tube should be allowed to remain during and after the operation.

OPERATION

Operation accomplishes three objectives, (1) removal of cause, (2) relief of intra-abdominal tension which has been favoring absorption, (3) removal of toxic material.

Operation will prove successful in a larger number of cases if it is short, if there is no evisceration and if the organs and intestines are exposed and handled as little as possible. Manipulations should always be gently performed.

The location for the incision is determined by the symptoms preceding peritonitis whenever this aid is available and properly interpreted. If no definite indications have been discovered a right paramedian incision is usually chosen. The primary lesion is sought, the area dried and walled off, and the perforation repaired. If the primary focus is the appendix this organ is removed, for the original perforation wherever it exists must be closed or the operation is worthless. It is advisable and permissible to remove as much free fluid as possible by absorbing it with damp sponges or by the use of suction tubes. The latter method is preferable, as it is possible to search all pockets with a minimum of peritoneal injury. Special attention should be paid to the hepatorenal pouch, the under surface of the diaphragm, and the pelvis. Unless the infection has existed over twenty-four hours the intervening areas may be neglected. If the fluid contains foreign material, such as food, fecal matter or calculi it is obvious that this must be removed. It should be borne in mind that the exudate in the immediate region of the perforation is more infectious and it is always advisable to wall this area off before manipulations are undertaken. Adherent fibrin should not be disturbed and it is usually better not to disturb adhesions unless they are enclosing a pus pocket. Wash-

ing out the abdominal cavity with water or salt solution only serves to distribute the infection more completely to all parts of the peritoneum.

Frequently one must decide whether immediate enterostomy should be performed. It is easier to do it before the abdomen is closed and one is more certain of utilizing a loop of intestine in the upper ileum or lower jejunum, rather than one too low or too high. If distention has already developed it is advisable to perform the enterostomy while the abdomen is open.

The question of drainage is debatable only in the first few hours, during the stage of peritoneal *irritation*. After suppuration is established, drainage must be introduced. The only objections to drainage seem to be the fear of causing adhesions which produce intestinal obstruction, the prolongation of recovery and the possibility of subsequent hernia. I believe that prolongation of convalescence is preferable to a postoperative course shortened by death, and all herniae may be repaired. The proper placing of drains will greatly reduce both the danger of troublesome adhesions and postoperative hernia.

Multiple drainage is objectionable and usually unnecessary. One pelvic drain may be sufficient, or one to the pelvis and one to the flank. The objections to drainage are best overcome by draining through stab wounds instead of through the incision, and the drains should be so placed that they lie along the parietal peritoneum instead of passing through coils of intestine. The stab wounds through which the tubes emerge are placed at the periphery of the abdominal plane. The common type of tubing used for drainage is too rigid and is sometimes responsible for necrosis. Cigarette drains, and all gauze drains for that matter, are nearly worthless because they become saturated in a short time and cease to function effectively. At the Elliot Hospital we have been using for a long time what we consider to be the ideal drain. It is thin walled tubing of nearly pure rubber, so thin that it will flatten out under light pressure. Through this are introduced two strands of ½ inch or 1 inch coarse meshed selvedged tape. This tape prevents collapse of the tube, furnishes some capillary drainage and allows ample tube drainage because the two strands of tape do not entirely fill the tube.

After drains are placed the incision is closed, with one or two small rubber tube drains carried to the tissues between the skin and the peritoneum.

POSTOPERATIVE CARE

Not infrequently the test of successful treatment is during the postoperative stage. While the type of treatment depends much on the condition preceding perforation, the extent and degree of peritonitis and the reaction of the patient to the infection, there are definite objectives to be attained in every case. The body heat must be maintained, the body supplied with fluids containing chlorides, the stomach must be relieved of retrograde toxic fluid, and fuel for metabolism supplied. Morphine should be used freely as it quiets peristalsis, prevents gas pains, relaxes muscles and induces quiet and rest. The patient should always be in Fowler's position.

External heat is always indicated after operation and the use of a lightweight electric pad over the abdomen aids circulatory activity. Introduction of fluid into the body must be instituted at once. Unless there has been perforation below the ileocecal valve, saline solution containing 5% glucose should be introduced into the rectum by the Murphy drip, by instillation, or by retained enemata. Fluid should not be given by mouth at first and not at all if nausea or vomiting is present. Hypodermoclysis of physiologic saline solution, either at intervals or continuously, should augment the rectal administration. Continuous hypodermoclysis is an admirable procedure, as suggested by Kanavel. Two No. 20 gauge needles are introduced, each joined by rubber tubing to a Y connection through which the fluid is supplied from a reservoir. If one area becomes uncomfortably filled, the flow may be stopped temporarily. In the more desperate cases, and where large amounts of fluid are needed immediately, and to reinforce methods described above, saline solution, physiologic or 1%, with or without the addition of glucose may be given intravenously. I use this method frequently, giving two to three litres at a time. The glucose addition is easily made by filling a 20 c.c. syringe with glucose and introducing it very slowly into the saline solution after puncturing the rubber tubing with the syringe needle. Judgment as to the amount of fluids necessary in a given case and the method of giving depend on the condition of the patient and the experience of the surgeon.

Continuous intravenous infusion of saline solution and glucose has been recommended because it is possible to introduce 3000 to 4000 c.c. of fluid daily for a period of several days. The necessary apparatus includes a reservoir for the fluid and a rubber tubing interrupted by a rectal drip bulb, above which is a compression clamp to regulate the flow. A connecting tip at the lower end of the tube fits the needle or canula which is inserted into an arm vein. The needle is fastened to the arm to prevent its displacement and the arm is wrapped about with a pillow. The rate of flow should be about 30 to 40 drops a minute. This method functioned with extremely good results in one of our recent cases.

The two most distressing symptoms which

may develop during the postoperative period are vomiting and ileus, either paralytic or mechanical. Vomiting may occur from toxic poisoning but usually is dependent upon a varying degree of ileus. For a period after operation the patient can ordinarily take fluids, though the oral intake should always be restricted. Vomiting usually commences in 24 to 36 hours, though it may appear later. At this time it will be noted that some distention has developed, especially in the upper half of the abdomen. As a rule the amount of vomitus is small and is not repeated for a few hours. The first vomitus is green and sour, but as repetitions occur, the fluid becomes darker and the odor more foul. Subsequently vomiting is repeated with greater frequency, and the disturbance becomes more marked, the patient regurgitating large amounts of dark brown fluid with a stercoraceous odor. Unless relieved, the condition of the patient from this time on is pitiable. Ileus has developed and there is a retrograde flow of intestinal fluid through the pylorus into the stomach, the stomach becoming dilated and vomiting occurring frequently.

The symptoms described above should not be allowed to occur if the indications are rightly interpreted when vomiting commences. Stomach washings with the gastric tube will relieve the patient, but the *early* employment of the Levin gastroduodenal tube No. 14 or No. 16 Fr. solves the problem in a much easier and more satisfactory way. This tube has a catheter tip and there are four oval openings within 6 cm. of the tip. It is easily introduced through one nostril into the pharynx, esophagus and stomach. With a syringe the stomach is washed out until the remaining fluid is fairly clear and free from mucus. The tube is fastened in a comfortable position to the side of the face and the patient is allowed to drink water or bicarbonate of soda solution at intervals. This washes out the stomach, and the privilege of drinking greatly improves the morale and comfort of the patient. In my experience I know of no one procedure that will do so much to improve the condition and promote the contentment of these desperately ill patients than the treatment just described.

Occasional use of the syringe must be resorted to, either to wash the stomach or dislodge mucus which is obstructing the openings. The tube must be allowed to remain until the stomach resumes its normal function. A careful record is kept of the oral intake and the outflow through the tube. In favorable cases it will be noted that more and more fluid is retained, until finally almost no fluid drips from the tube. As the patient improves the external end of the tube is gradually raised above the level of the stomach. During the period when the stomach is not functioning, saline solution and glucose must be administered intravenously or subcutaneously.

While the tube treatment has been indicated for vomiting, it is also the best treatment for paralytic ileus. There is a diversity of opinion regarding the effectiveness of drugs such as pituitrin and eserin. It is probable that they have no effect until such time as the paralysis abates and the muscle tissue begins to resume its tone. If they are physiologically effective they are obviously detrimental in mechanical ileus. It must be admitted that in paralytic ileus both drugs *seem* to produce results occasionally.

Whenever vomiting or symptoms of ileus develop it is well to commence the use of evacuating enemata. These should be given at intervals of four to six hours depending upon the condition and progress of the patient. I prefer to use four ounces of glycerine for this purpose because it is as effective as any other agent, is tolerated well by the rectum and is not so uncomfortable as the bulkier mixtures.

It has been suggested that the use of the Levin tube, by relieving symptoms, would obscure a diagnosis of mechanical ileus. As a matter of fact, the use of the tube aids in the diagnosis if the reflux from the stomach is watched in conjunction with the observation of the general symptoms of the patient. When the returned fluid continues to remain stercoraceous after twelve hours, mechanical obstruction should be suspected.

Mechanical ileus, as a complication of peritonitis, must be treated surgically and recognized as early as possible. Operation should be performed whenever there is a reasonable suspicion of obstruction. If one waits for all the classical symptoms of obstruction to develop, it is then too late to benefit the patient by operation. The chief and most perplexing difficulty is in differentiating paralytic ileus and actual obstruction. If operation is decided upon, the abdomen should be opened through a new incision, the obstruction found and relieved, if possible, and with minimum manipulation. Enterostomy is also advisable. If the obstruction cannot be located an enterostomy or a cecostomy must be performed as this is the only procedure that will give relief, and it may save the life of the patient.

The group of symptoms and the complications associated with peritonitis present one of the most trying and difficult tasks with which the surgeon has to contend, and one is indeed fortunate when success crowns the long hours of thought and effort required to carry the patient through this ordeal to recovery.

SUMMARY

The size and location of the perforation may determine the extent and severity of peritonitis.

Operations performed during the stage of irritation are invariably more successful than those performed after the suppurative process has developed.

Symptoms usually are very definite and diagnosis is made from history and physical examination. Rarely is it necessary or advisable to wait for laboratory corroboration.

All patients will die without operation.

Administration of saline solution is advisable before, during and after operation.

Operation should be brief and add as little as possible to the patient's shock.

Drainage is always safer, but the use of many drains is unnecessary. Location is more important.

Enterostomy is frequently advisable.

The use of continual stomach drainage is urged as soon as a tendency to vomiting occurs.

A method of stomach drainage is described which gives immeasurable relief to the patient.

Several methods of supplying saline solution with glucose either continuously or intermittently are described.

It is necessary to supply large amounts of fluids.

Drugs are of little or no benefit in the treatment of ileus. Constant drainage of the stomach with an inlying tube is always indicated. The use of frequent enemata is advised.

Relief of mechanical ileus is surgical, and operation must be instituted as soon as indications point to definite obstruction.

Discussion

Dr. James W. Jameson, Concord: Dr. Wilkins is to be congratulated on the paper which he has delivered, not only because of the form of delivery and its subject, but also because peritonitis is a very important factor in general surgery and one which, unless properly treated, is usually fatal.

The age of the patient has an important bearing on the prognosis of cases suffering from peritonitis, and it is well-known that children do much better than elderly patients.

Peritonitis following the sudden rupture or perforation of a viscus is apt to be more rapidly fatal than that resulting from a slow perforation. This is no doubt due to an overwhelming bacterial invasion or to chemical irritation in addition to the infecting organism, against which the peritoneum is unable to react. In the slower perforation, systemic resistance may be aided by the development of antibodies, and local inflammation tends to form protective adhesions. Pain in the former case is more intense following perforation and in the latter is often followed by relief, the pain gradually becoming worse as the peritonitis extends. This temporary relief may be interpreted as an improvement in the patient's condition and may result in a delay in operation. In the case of extensive peritonitis with persistent vomiting and extreme distention, the Levin tube which Dr. Wilkins speaks of is of great help, and supplementing that, I think colon irrigations with warm solution are very beneficial.

I cannot agree with Dr. Wilkins regarding drainage, for I think a rubber tube should be used only to keep the abdominal wall open and should extend just through the peritoneum, otherwise injury from pressure will result. We know that the peritoneal cavity cannot be drained and that the infective material will follow the line of least resistance; consequently if we introduce two pieces of small Penrose tubing into the infected area and keep the abdominal wound open with a tube we will accomplish all that is possible.

Dr. Chester L. Smart, Laconia: I feel as I knew I would when I get up to discuss a paper written by Dr. Wilkins and discussed by Dr. Jameson. There is little left for me to say.

In the first place I want to emphasize what Dr. Jameson said about the excellence of Dr. Wilkins' paper. It recommends some definite things for us to do and I think few of us could take exception to any of it.

Two points have not been emphasized: First, the resistance of the patient and second, the virulence of the infecting organism. I recently had in the hospital a case of general peritonitis which illustrates this particular phase.

A woman, 35 years old, the mother of eight children, the youngest one two months old was taken Friday, May 1, with an abdominal pain, and, acting on her own initiative, took a dose of salts. Saturday she was vomiting and also had profuse diarrhea. She was seen by a doctor that night; the temperature was 101°, the pulse 120, and there was some distention with no localized symptoms.

Sunday morning the pulse was 130, the temperature 102° and she was vomiting. On Sunday night there was much distention, with a pulse of 135, a temperature of 103° and rapid respiration. She was brought to the hospital on Monday, May 4.

This is the chart of the situation on admission. I will not go into details of the treatment except to emphasize the amount of saline solution given both rectally and by hypodermoclysis. It was a question of whether we should operate. We did and found an abdomen full of pus and the appendix perforated and floating about.

A Penrose tube was put in at the appendix region and another through a stab wound above the pubis. At the time of operation, her temperature was around 105-104.6°, pulse 140; next morning her temperature was 100°, pulse 120 and respirations 30. The first day after the

operation there was much distention; 2000 c.c. normal saline solution were given subcutaneously. The second day her abdomen was distended; 2000 c.c. saline solution with glucose were given and this was repeated daily. The fourth day there was free evacuation of the bowels and much less distention. The seventh and eighth days her temperature was normal, there was no vomiting, but the pulse was 130 and at no time was below 120. She went along until the tenth day and then became restless, with pulse between 130-140, abdomen not distended and bowels moving with a free discharge of pus through abdominal drains. She died on the twelfth day.

It would seem in this instance that the woman died, not of peritonitis but of a toxic myocarditis due to absorption and that the fact that she was a young woman, mother of eight children, the youngest two months old, was a real factor in lowering her resistance to a virulent infection.

Dr. F. B. Lund, Boston, Mass.: I agree with the other speakers and congratulate Dr. Wilkins on his very sensible and thorough paper. One thing has been said, that it is impossible to drain the peritoneal cavity. That statement was made by the late Dr. David W. Cheever in 1889. It is perfectly true, but we can at least drain it temporarily until it becomes straightened out. Some twenty years ago I went to New Orleans and reported three cases of enterostomy for peritonitis after an operation for acute appendicitis, in which the patients recovered. Since that time most of the enterostomy patients have died, and the reason is that we are doing this operation in cases where the patient is sicker from the toxemia than from the obstruction, and the intestines are adherent. Dr. Wilkins did not say any too much about the duodenal tube; it has been a life-saver. You know how infection progresses rapidly in some and slowly in others. The bacteria vary in toxicity, and if active are apt to get ahead in spite of anything you can do. In my experience with these people who get distention and vomiting, if the pulse is going down and the temperature also, the condition will improve. After trying everything, including the tube, they may still continue to distend and then all of a sudden throw up a lot of stomach contents and get well. These are the ones that are suffering from paralysis rather than from toxemia. Instead of enterostomy I am now willing to wait and try the duodenal tube. There is so much to think about in this connection that perhaps I had better not say anything more except to congratulate the doctor upon his paper.

Dr. George S. Foster, Manchester: I want to say a word about the abdominal wall in these cases. I have been using, over a long period of years, a fortified solution of boric acid for fomentations. I think in the average case we get distention and this treatment tends to reduce the distention. We are also apt to get secondary infection of the wall and these fomentations rather assist in fortifying the wall. I feel that with patients suffering from general peritonitis, or even with localized peritonitis, we sometimes can do too much at the time of operation. I think that if we do just enough to free any confined pus;—turn the patient on the side where the putrid material has accumulated so that it will drain out, and not do too much after the operation, they stand a better chance to get well.

Dr. Frederick P. Scribner, Manchester: The reduction of blood pressure can be controlled in most cases by the use of ephedrine sulph. hypodermically in doses from ¾ gr. to 1½ grains. I use this routinely in all my spinal cases. If we later get too great a drop in the blood pressure, we use adrenalin in 10 m. doses, hypodermically. As to the position of the patient, in these cases of a ruptured viscus, I also have felt that it would be preferable to have the head raised rather than lowered, but on talking with Dr. Babcock and Dr. Pitkin I found that they disregard this feature, because of the many other advantages of spinal anesthesia.

MISCELLANY

A CORRECTION

The following corrected discussion of the paper on Coronary Occlusion and Angina Pectoris by George Blumer, M.D., appearing in the New Hampshire Section, September 3, 1931, page 497, has been forwarded by Dr. Thomas M. Dudley of Concord, N. H.:

The first case I have in mind is a man of 52 years. His complaint was pain in both arms with substernal oppression. Observation at home under nitroglycerine, bromides and rest showed no improvement. Physically he was thin, apathetic, had a slightly enlarged thyroid, an enlarged heart, tachycardia, but no congestive failure, his blood pressure was 170/90. Hospital study showed a B.M.R. of plus 30. Lugol's Solution caused distinct improvement and he was discharged for observation. In a month the angina was again uncontrolled and operation decided on. A hemithyroidectomy was done, the gland being much larger than at first suspected and very glandular and turgid. Convalescence was slow, the angina was benefited, but hyperthyroidism persisted and he died about three weeks after operation from auricular flutter.

The second case is one of diabetes mellitus in a 60-year-old librarian. After insulin his angina was markedly improved but in several months recurred with renewed vigor. At this time he was found to be quite anemic but apparently from a secondary anemia. A second remission of symptoms occurred

with rest but in 3 months he was again incapacitated by angina. This time clinicians in another city arrived at a diagnosis of pernicious anemia. With liver therapy his attacks have entirely disappeared.

Let me reiterate that a diagnosis of angina alone does not convict a man to the shackles of invalidism. We must look further since the rectification of some underlying chronic disease may open new avenues for the relief of anginal symptoms.

NEW HAMPSHIRE TUBERCULOSIS LEAGUE

Dr. Edward O. Otis, honorary president of the Massachusetts Tuberculosis League, will be among the honor guests at the annual meeting of the New Hampshire Tuberculosis Association at the Eagle Hotel, Concord, N. H., on October 1.

Guest speakers will be Hon. John G. Winant, Governor of New Hampshire, Dr. H. E. Kleinschmidt of New York, Director Medical Service, National Tuberculosis Association, and Mr. Frank Kiernan, Executive Secretary of the Massachusetts Tuberculosis League.

Governor Winant, president of the Association, will preside. Mayor Robert Brown of Concord will felicitate the gathering on behalf of the city, and Dr. Robert B. Kerr, executive secretary of the New Hampshire Tuberculosis Association, will discuss the high lights in the year's campaign against tuberculosis in New Hampshire. Officers will be elected for the ensuing year. Dr. Kleinschmidt's subject will be "Adventures in Case Finding."

A BEQUEST TO THE PORTSMOUTH HOSPITAL

By the will of Miss Eleanor B. Haven of Lancaster, Mass., the Portsmouth Hospital of Portsmouth, New Hampshire, will receive one thousand doNars.

SUMMARY OF THE MIDDLE-RATE PLAN FOR HOSPITAL PATIENTS — THE FIRST YEAR'S EXPERIENCE OF THE BAKER MEMORIAL OF THE MASSACHUSETTS GENERAL HOSPITAL, BOSTON, MASSACHUSETTS

(Continued from page 678)

the agreed fee to the physician who has been in charge of the patient.

This middle-rate plan has been designed to meet the needs of patients who cannot meet the costs of charges and fees in the private room pavilion of the hospital, yet who are able and anxious to pay their sickness bills on an independent basis, rather than accept care in a ward where the patients' payments cover only half the hospital's costs and where the doctors receive no remuneration. The Julius Rosenwald Fund is coöperating by an agreement to meet half the deficit to be anticipated in the first three experimental years. Once fully established, the service is expected to be self-supporting.

The Baker Memorial plan is similar in aim and provision to that recently tried in the hospitals of Keokuk, Iowa, in coöperation with the Julius Rosenwald Fund, and discontinued after a year because of the opposition of some of the Keokuk doctors. In Boston there has been no medical opposition. The 200 staff physicians at the Massachusetts General Hospital have given complete coöperation. Of these, 183 have served patients under the plan. Payments by patients to physicians during the year covered by the report were approximately $147,000. While a few of these 2,500 patients might have found it possible to obtain care in the regular private room service, the majority would have been obliged to seek ward care had not the middle-rate plan been in operation, and the physician would then have received no fee at all.

The total costs paid by patients for their illness under the middle-rate plan averaged $173, the average length of stay in the hospital being 13 days. The average was made higher by a few instances of very prolonged illness, one requiring more than six months' care and a bill of $1,769. "Hospitalized illnesses are high-cost illnesses, even under the best of conditions," Dr. C. Rufus Rorem, author of the report, comments. "Fully half of the illnesses, however, cost not over $118. Such an expenditure is a large item in a family budget of $2,500 a year and it is a still greater percentage of the smaller budgets . . . which existed in some cases. . . . The care of a hospitalized illness, therefore, must be regarded as a major item in the expenses of the fiscal period in which it occurs. Any device which can limit the necessary costs must be considered important."

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 17401

FATAL HEMOPTYSIS

SURGICAL DEPARTMENT

PRESENTATION OF CASE

DR. RICHARD W. DWIGHT*: This patient was a thirty-four year old American who came into the hospital in the beginning of November.

He had a history of having been well until a year previous to that time, when he had all his teeth removed under ether anesthesia. He stayed in bed one day, went back to work the following day and felt well. Three weeks after that he had what was diagnosed as pneumonia in the right chest. He was treated by his own doctor at home and was in bed for about three weeks. He also had pleurisy in the right chest. One chest tap was done, at which about an ounce of fluid was removed. He did not know what the fluid was like. Towards the end, about three weeks after the onset of the pneumonia, he had a severe fit of coughing and raised a great deal, he thought a quart or more, of foul greenish sputum. It was the first time that his sputum had ever been very large in amount. After he got over the pneumonia he was able to go back to work, but did not feel well. He continued his usual work as a milkman until about a month before he came to the hospital. He had continued cough all this time, coughing most just as he was going to bed at night and when he first got up in the morning. The sputum was not blood stained, but it was very foul, so foul that at times it nauseated him. He did not know how much there was; he never measured it. After he got over the pleurisy which he had had at the time of the pneumonia he had no pain at all, but more severe fits of coughing, giving a great deal of soreness under the sternum. He continued at his work. About three months before he came into the hospital he had severe night sweats every night for a month. Apparently after that his work was lightened somewhat and he had no more night sweats. He got out of breath more easily than formerly, but could climb a flight of stairs without difficulty. His doctor advised postural drainage, which he carried out three times a week, leaning over the side of the bed, but he never noticed any relief from it.

*Recently senior interne on the West Surgical service.

The rest of the history is essentially negative. He had an infected sinus which was treated at the Eye and Ear Infirmary about three years ago at the same time his nasal septum was straightened. Two years ago he had tonsillectomy and adenoidectomy at the Eye and Ear Infirmary.

Physical examination when he came in showed a fairly well developed, rather thin man about thirty-four, quite anemic looking, coughing frequently and raising small amounts of foul sputum. The chest was clear in front. In the back there was dullness on the right side extending upward a little over the angle of the scapula, with very much diminished breathing and practically absent tactile fremitus. There was some egophony above the dullness.

The red blood cell count was 4,000,000 and the white cell count 15,000.

He was running a slightly irregular temperature going up to 100° every night, occasionally a little higher.

He was taken to the Eye and Ear Infirmary, where bronchoscopy was done. About an ounce of very foul pus was found in the trachea and two ounces in the right main bronchus, and much was coming out from the stem bronchus to the right lower lobe, the middle terminal bronchus of the lower lobe and the slit-like bronchus laterally.

He came back to the Massachusetts General Hospital. About the end of November he had a severe hemoptysis of about 16 ounces of blood. The red blood count dropped to 2,000,000 and he was transfused. Shortly after that he had a phrenicectomy on the right side. He improved somewhat after that and coughed less. Through December he continued to run an irregular temperature, 102° at times. He was up part of the time, but most of the time was in bed. At the end of December he had another fairly severe hemoptysis and had to be transfused again. His chart looked worse all the time. Gradually the swings became bigger and bigger and he seemed to be losing ground. Apparently there was nothing that could be done surgically. He continued in the same way through January, having small hemoptyses of one or two ounces every five or six days. On the thirtieth of January he had a severe coughing attack and massive hemoptysis and became cyanotic. He was given oxygen and stimulation, but died within half an hour.

He had numerous x-rays while in the hospital.

DR. GEORGE W. HOLMES: The earliest film we have shows a process apparently occupying the region of the right lower lobe. There are small bright areas in it which might be dilated bronchi. At the apex the process is not sharp and looks as though it were infiltrating the lung. You can see that the outline of the diaphragm and the costophrenic sinuses are obliterated. It is the appearance of a process infiltrating the lower lobe rather than fluid or encapsulated pus.

The heart is not displaced in that picture, which would be against plugging of the bronchus.

There are a number of films in between, but I have selected the ones which seem to show a definite change. Here is a film showing a process in the upper part of the chest as well as one in the lower part. In the oblique and the direct lateral views you can see the middle lobe very distinctly. The process involves the lower part of the upper lobe and the whole of the lower lobe. There is a dull area which may represent the interlobar septum. There is a collection of fluid between the lobes.

X-RAY INTERPRETATIONS

November 15. The appearance is that of consolidation. It may well be an abscess, but no cavity can be demonstrated.

December 30. Consolidation in the right upper and lower lobes.

January 13. Multiple abscess cavities.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Lung abscess.

ANATOMIC DIAGNOSES

Multiple abscesses of the right lung.
Edema of the left lung.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: At autopsy we found a very extensive process throughout the right lung, a whole series of abscesses. Practically the entire lower lobe was involved, and also the posterior half of the middle lobe and the lower third of the upper lobe. The interlobar septum was almost entirely destroyed. Presumably the process extended directly through it. The pleural cavity on that side of course was almost totally obliterated. The abscess cavities varied from one centimeter and a half to nearly four centimeters in size. The majority of them contained blood, undoubtedly a terminal, fatal hemorrhage. A few had purulent exudate in them. The blood had not been aspirated into the normal lung as it often is. The left lung showed only a moderate edema.

The rest of the autopsy was essentially negative.

Microscopic examination shows in the immediate neighborhood of many of the abscesses blood vessels partially or completely thrombosed. The process represents a race between the invading infectious process and the protective mechanism of thrombosis. In one or more areas the infection evidently got a bit ahead of the protective mechanism, with a fatal hemorrhage as the result.

Most lung abscesses that we see die of one of three things, (1) a fatal hemoptysis, as in this case, (2) extension of the infectious process to the other lung with rapidly spreading septic pneumonia, or (3) penetration into the pleural cavity with empyema.

DR. DWIGHT: Was the pleural cavity pretty well obliterated?

DR. MALLORY: Almost wholly. Over the anterior margin of the upper and middle lobes there was still a little pleural cavity left. The remainder was entirely obliterated.

A PHYSICIAN: Were spirochete stains done in this case?

DR. DWIGHT: Yes; numerous spirochete stains were done and many spirochetes were found.

DR. HOLMES: Was there any evidence to show that the middle lobe was involved after the upper, or do you think the process extended from the lower lobe to the middle lobe and then up?

DR. MALLORY: I do not believe I can tell, Dr. Holmes.

DR. HOLMES: I was just wondering whether my interpretations were right.

DR. MALLORY: I think very probably they are. But the process had gone so far at the time of autopsy that it would be impossible to tell which is the oldest portion.

CASE 17402

PATHOLOGIC FRACTURE OF THE FEMUR

SURGICAL DEPARTMENT

PRESENTATION OF CASE

DR. VERNON P. WILLIAMS*: *First admission.* This patient was a widow of seventy-three who first came into the hospital in March, five months before her second admission. At that time her complaint was of a lump in her left breast, together with a prickling sensation in the breast of six weeks' duration. Except for these she was perfectly well.

The past history is irrelevant except that about three years previously she severely injured her left breast and also broke her left humerus in four places.

When she was examined she was found to have in the left breast a movable, irregular, very firm mass which seemed not to be attached to the tissues beneath. In the left axilla was a very firm gland the size of a cherry. Moderate hypertension was also noticed, and procidentia.

A chest film taken upon entry showed no evidence of metastasis in the chest or the cervical spine.

She was put down as being a poor ether risk. However, after a preparation with sodium bromide she was operated upon five days after entry. A radical mastectomy was done. After the operation she made a satisfactory recovery and left the hospital about three weeks after admission.

History of interval. Soon after going home she began to have what she called "neuritis",

*Recently senior interne on the East Surgical service.

—weakness and pain in all the extremities, until because of this weakness and pain she decided to use crutches to help her in getting about the house. The day before she reëntered, four months after her previous discharge, she fell and suffered apparently a fracture of the right femur.

Second admission. When she was brought into the hospital for the second time we found on examination that she had a fracture in the midshaft of the right femur. X-rays could not establish definitely that it was a pathologic fracture, although that seemed probable. However, in the femur below the fracture line there were several areas of diminished density, unquestionably metastases.

Physical examination at this second entry showed also what was apparently a recurrence of the carcinoma in the mastectomy scar. It was also noticed that her right upper arm was weak and the muscles of that part were somewhat atrophied.

X-rays were taken at various parts of the body and metastases were shown scattered throughout the skull and also in some of the ribs on the left side and perhaps in the cervical spine, although that was not observed definitely.

Because of her condition and the hopeless prognosis the fracture was treated simply with skin traction. She went along until seventeen days after entry without any difficulty except that she had residual urine and had to be put on constant drainage. Then her temperature went up to 102°, her respirations increased and she was slightly cyanotic. A medical consultant's diagnosis was pneumonia of the right lung. We gave her what supportive treatment we could. The white cell count was 19,000. She sank rapidly. The next day her temperature went higher and her chest filled with râles. She died that afternoon, that is about seven months after the first symptom.

Dr. Ernest M. Daland: I should like to ask what x-rays were taken before her operation.

Dr. Williams: Only a chest film and the lower cervical vertebrae.

Clinical Discussion

Dr. Daland: There was nothing of any particular importance about the first examination. It was an advanced lesion with a definite axillary gland. She was a poor risk, but stood the operation fairly well and made a good convalescence.

The night before her second admission her doctor called me up and told me he thought she probably had cerebral metastasis because she showed weakness in her right arm and general weakness in the right leg too. He also noticed recurrence in the scar. He asked me to come out and see her the next afternoon. But early the next morning he called me and told me she had fallen and sustained a fracture of the femur.

The important lesson here is that we do not know positively that her bones were free from disease at the time of the first operation. I am more and more impressed with the necessity of x-raying the spine, the pelvis and the long bones as well as the chest before doing radical surgery, because many bone metastases are picked up by routine examination. The chief reason we have not done it more in this hospital is because of the expense to the patient. The cost of eight or ten x-rays adds a good deal to the bill of a patient who is having trouble in paying board.

So far as treatment of the fracture went, that was purely a palliative procedure. We put her on skin traction without much weight in order to reduce the muscle spasm and make her as comfortable as possible. We knew at that time that she did have metastases in the cervical spine, recurrence in the scar and probably metastases in her lung, so we had no hope of getting her on her feet. In some instances a pathologic fracture put up in traction in this way does get bony union. There are several cases in which a patient has gone on for a period of five or six months with union enough so that he can move about in bed without any other appliance and without any discomfort. These patients usually sustain a fracture at the same site or a different site later on. I think general metastases in other places than the site of a pathologic fracture kill the patient more commonly.

Dr. George W. Holmes: This chest film shows first a very high diaphragm on the right side. That may be due to involvement of the liver, which has not been mentioned. It might be due to masses of glands at the root of the lung pressing on the bronchus and preventing normal flow of air into the lung. Those nodules are not metastatic nodules. One could see as much as that in any older person who had a lung infection. From the x-rays alone I do not think one could make a diagnosis of metastasis to the lung.

I should like to ask Dr. Daland a question, if I may, in regard to the healing of fractures in metastases from carcinoma. Did I understand you to say that you get union in some cases where there was metastasis from carcinoma of the breast?

Dr. Daland: Yes. I have seen three cases in which we got union so that the patients could move from side to side in bed without an appliance and without pain.

Dr. Holmes: We do not seem to get much callus in the treated cases, and I am wondering if they do not do better without treatment. Perhaps treatment prevents the formation of callus.

I should like also to emphasize the necessity of taking films of the pelvis and long bones. We may have extensive metastases in the bones and no metastases in the lungs. When the pelvis is negative the long bones are not likely to be involved. So that if we made it a matter of routine to take films of the chest and the pelvis we should not miss many cases.

DR. DALAND: For the past three years at the Pondville Hospital we have taken routine x-rays of the chest, spine, pelvis, femora and humeri on all patients entering the hospital with carcinoma of the breast, either before or after operation. We have picked up a few cases with bone metastasis that otherwise would have been operable.

Recently Dr. Dresser and I decided that we might cut down on the number of x-rays taken in these cases, for it seemed that if the lungs, ribs, spine and pelvis were negative there was practically no chance of finding disease in the long bones, and it was decided that where the former pictures were negative the long bones should not be x-rayed. Within one week we accidently found an extensive metastasis in a humerus shown on the chest film where all other bones and the chest were negative. This disproved our previous theory, and we have returned to the routine examination by x-rays of all bones except those below the elbow and the knee.

DR. TRACY B. MALLORY: We have been interested in checking that to a slight extent here, and on the whole it seems to hold fairly well. There are numerous exceptions of course, as one would expect, but in general two-thirds of the cases seem to follow that rule. We went back over the slides in this case after the autopsy and found that we had graded it as a II at the time of the original breast operation. I am inclined now to think that we were not correct in that, and that it is a III.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Carcinoma of breast.
Metastases to femur. (Pathologic fracture.)

ANATOMIC DIAGNOSES

(Adenocarcinoma of the breast.)
Metastatic adenocarcinoma in the lungs, the liver and the femur.
Pathologic fracture of femur.
Traumatic fracture of third left rib.
Bronchopneumonia.
Scar of operation wound, left mastectomy.

PATHOLOGIC DISCUSSION

DR. MALLORY: At autopsy we found metastases in the bones, the lungs, and also in the liver. I have a section from the original tumor and also one from the tumor in the femur which was responsible for the pathologic fracture. The latter shows marked secondary bone formation, which as you know is common in these metastatic lesions from breast cancers. Some types of metastatic tumors, the majority perhaps, produce primary destruction in the bone. But two types occasionally lead to a good deal of bone formation as well as destruction, breasts and prostates being the two of which this is most characteristic.

The first slide is from the breast cancer. This is a typical scirrhous process with marked invasion at the periphery. There is a trace of gland formation, but not a great deal. The mitotic figures are fairly numerous. I think on review of the slides I should be inclined to grade it as a III.

Have you had enough experience, Dr. Daland, with other types of bone metastases to know whether those from breast cancers are more apt to heal than metastases from hypernephroma, for instance?

DR. DALAND: I do not wish to give the impression that all fractured femurs with metastases from cancer of the breast heal. I think they are a little more apt to than those from other tumors. Yet I have seen a great many more which have not shown any signs of healing. But I am impressed with those first three cases I had when I was first in practice on cases no one else wanted to take care of and turned over to me.

DR. MALLORY: I think it is quite reasonable to assume that the type of tumor in which one gets a great deal of secondary bone formation would be more apt to heal than the primarily osteolytic lesions.

DR. HOLMES: I have two cases which I am treating now. One was metastasis from the breast. Both grow in spite of radiation. As I said, it may be that radiation destroys callus. It is quite important to decide whether to treat them or not. This case showed new bone formation very clearly.

DR. DALAND: I have heard Dr. Morrison say that it is better to undertreat them than to overtreat them.

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MENINGOCOCCUS MENINGITIS

MENINGOCOCCUS meningitis is a baffling disease. We know that it is infectious and that healthy persons may carry virulent meningococci in the nasopharynx, but the reason why, in interepidemic periods, sporadic cases fail to give rise to localized outbreaks, and why in epidemics the disease spreads so rapidly is beyond our ken. We know that the meningococcus probably enters the body through the nose and frequently invades the blood stream before any symptoms of meningeal infection appear, and that sometimes these cocci remain localized in the blood. The pathology of meningitis is its best understood feature.

The discovery of an antimeningococcic serum by Flexner in 1907 did much to rob this disease of some of its terrors. Through the intelligent use of specific serum the fatality can be reduced from the usual seventy to eighty per cent. to about twenty per cent., but often in spite of early diagnosis and proper serum treatment death may occur. There are several causes for such failures. The infecting strain of meningococcus may be of such an exalted virulence that no treatment will avail. The peculiarities of the anatomy of the subarachnoid space, particularly in infants, may cause block and prevent the diffusion of the injected serum. In skilful hands this difficulty may be overcome by ventricular or cisternal puncture and the administration of serum by these routes, as well as by the spinal canal. A further complication may be that the serum, immunologically, does not "fit" the particular strain of meningococcus infecting the patient.

Previous to the development of serologic methods, all meningococci were supposed to be biologically more or less alike. Then Dopter, Flexner and others found that through their serologic reactions the various strains could be divided into two, and later three groups. Subsequently Gordon and Murray made a reclassification and established four groups. As a result of this knowledge, serum manufacturers have used strains of all four groups in the immunization of horses for the production of a specific antimeningococcic serum. While this serum may be therapeutically efficacious in the majority of cases, it is known that different lots of serum may vary in their potency for this or that strain of meningococcus, and that at times it may be necessary to determine the biologic identity of the infecting organism and to select a serum with a higher immunologic value for that particular strain.

Another reason for the curative failure of serum in some cases may be found in the studies on meningococcus reported by Butterfield and Neill, by Evans and still more recently by Branham, Taft and Carlin[1], all of the National Institute of Health. To the four established groups of meningococci, Branham adds another group consisting of strains isolated from patients in the recent mid-western outbreaks, which apparently bears little or no immunologic relationship to other known groups, and such strains, therefore, would not be covered by our present serum. A comparison of the work of these authors shows that the predominating group may vary from year to year; that while meningococci of a single group may be the cause of an isolated outbreak, in the more extensive epidemics all varieties may be involved. Another fact disclosed that is pertinent to our present discussion is that strains isolated from sporadic cases in interepidemic years appear to be aberrant strains, that is, they fall into none of the usual groups.

Complicated as the situation appears, whether viewed by the clinician, the epidemiologist or immunologist, there is no reason for despair. Early diagnosis, with prompt and proper serum treatment under bacteriologic control more frequently than not bring gratifying results in the saving of life and the prevention of distressing sequelae. The disease, its organism and its serum are all being subjected to renewed and extensive studies in the clinics and laboratories of

this country. New facts are being brought to light that must eventually result in the production of a more potent serum of wider immunologic coverage. This with the use of modern methods of its administration into cistern and ventricle, and the establishment of free subarachnoid irrigation and drainage, should bring to recovery many patients who would otherwise die.

The physicians of Massachusetts are fortunate in having at their command a special meningitis service offered by the State Department of Public Health. A consultant is at the call of any physician and will see, with the physician, suspected or diagnosed cases of meningitis; he will make spinal punctures, administer serum when indicated and carry out bacteriologic and serologic controls. This service[2], like the special service offered for years by Neal of New York City[3] is a valuable aid to the practicing physician, and already has shown gratifying results in lowering the fatality from this disease.

REFERENCES

1 Branham, Sara E., Taft, Clara E., Carlin, Sadie A.: Studies on meningococci isolated in the United States, 1928-1930. Public Health Reports, Vol. 46, No. 16, pp. 897-916, April 17, 1931.

2 Anderson, Gaylord W., and Bigelow, George H.: Epidemic meningitis control. New Eng. Jour. Med., Vol. 202, No. 11, pp. 518-520 and 546, Mar. 13, 1930.

3 Neal, Josephine B.: Service for meningococcic meningitis. New Eng. Jour. Med., Vol. 202, No. 18, pp. 859-863, May 1, 1930.

This service may be obtained, free of any expense to physician or patient by telephoning the State Department of Public Health (Haymarket 4600) or the Children's Hospital of Boston (Aspinwall 5930).

A NEW STATE MEDICAL JOURNAL

It is always a pleasure to welcome a newcomer to the ranks of the State Medical Journals. We have recently received the first number of the first volume of the new Journal of the Medical Association of the State of Alabama, the official organ of the Alabama State Medical Society and of the Alabama State Board of Health. This first issue contains the Annual Address of the President of the Association at its meeting in April, the Annual Jerome Cochran Lecture on Pathology of Tuberculosis and Syphilis, and a particularly interesting article by Dr. Weil of Montgomery on the Plants Causing Hay-Fever in Alabama. This last paper is accompanied by a Pollen Calendar for the State ranging from the Alder in January to the Giant Ragweed in September and October. The remainder of the issue includes editorials and miscellanea, especially the Annual Report of the Alabama State Board of Censors.

We congratulate the Editor on the attractive appearance of this first number of the new Alabama State Medical Journal for which we are glad to prognosticate a creditable and successful career of service.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Turner, George Grey. M.B., M.S., F.R.C.S. (Eng.) M.D. University of Durham, 1898. F.A.C.S. Professor of Surgery in the University of Durham. Senior Surgeon, Royal Victoria Infirmary, Newcastle-upon-Tyne. Member of Council of Royal College of Surgeons of England. His subject is: "Some Experiences in the Surgery of the Oesophagus." Page 657. Address: The Hawthorns, Osborne Road, Newcastle-upon-Tyne, England.

West, George Leon. A.M., M.D. Harvard University Medical School 1894. Visiting Physician, Newton Hospital. Medical Examiner, Seventh Middlesex District, Commonwealth of Massachusetts. His subject is: "Sudden Death in a Case of Asymptomatic Subacute Bacterial Endocarditis." Page 675. Address: 860 Beacon Street, Newton Centre, Mass.

Meaker, Samuel R. A.B., M.D. Harvard University Medical School 1915. F.A.C.S. Professor of Gynecology, Boston University School of Medicine. His subject is: "Pregnancy and Fibromyoma: Report of a Case Complicated by Rectal Obstruction." Page 679. Address: 475 Commonwealth Avenue, Boston.

Boland, Benedict F. B.A., M.D. Tufts College Medical School 1920. F.A.C.S. Junior Visiting Surgeon for Gynecology and Obstetrics, Boston City Hospital. Assistant in Urology, Massachusetts General Hospital. Assistant in Gynecology and Obstetrics, St. Elizabeth's Hospital. Instructor in Obstetrics, Tufts College Medical School. Address: 270 Commonwealth Avenue, Boston. Associated with him is:

Cochran, Williams. B.A., M.D. Harvard University Medical School 1929. Resident Pathologist, Boston City Hospital 1929-1930. Surgical Interne, Boston City Hospital. Address: Boston City Hospital, Boston. Their subject is: "Laboratory Study of the Gonococcus." Page 680.

Wilkins, George C. M.D. Harvard University Medical School 1899. F.A.C.S. Surgeon, Elliot Hospital, Manchester, N. H. Member, New Hampshire State Board of Health. His subject is: "Treatment of Peritonitis Resulting from Perforated Viscus." Page 682. Address: 814 Elm Street, Manchester, N. H.

The Massachusetts Medical Society

STATED MEETING OF THE COUNCIL

A stated meeting of the Council will be held in John Ware Hall, Boston Medical Library, on Wednesday, October 7, 1931, at 12 o'clock, noon.

Business:

1. Reading record of last meeting in abstract.
2. Report of Committee of Arrangements for Annual Meeting.
3. Reports of Committee on Membership and Finance.
4. Reports of committees to consider petitions for restoration to the privileges of fellowship. Appointment of a new committee.
5. Appointment of Auditing Committee to audit Treasurer's accounts as of December 31, 1931.
6. Incidental Business.

WALTER L. BURRAGE, *Secretary.*

Brookline, September 30, 1931.

MISCELLANY

LECTURES ON HEALTH PROTECTION

Starting Wednesday, October 7, at 8:00 o'clock, in the Boston English High School, Warren Avenue and Dartmouth Street, entrance on Warren Avenue, Boston, the first of a series of ten illustrated lectures on "Health Protection" will be given free by Julius Gilbert White, a nationally known authority on foods and nutrition. These lectures will be held on each successive Wednesday in the Boston English High School and are conducted under the auspices of the Health Education Association of which Mrs. Nellie A. Barrington is the director. Mr. Morris M. Goldberg, Manager of the English High Center, enthusiastically endorses and coöperates in this series of lectures.

This course explains the causes of disease and the principle by which one may live longer and better. The instruction includes correct nutrition, scientific cookery, good health habits, home care of the sick, hydrotherapy and other rational treatments and many other practical aids in the conquest of disease.

The purposes of these lectures are to aid the average person in simplifying eating, to assist in reducing food bills, and to build up a reserve of strength, health, and happiness. The course is constructed progressively—every lecture is based upon those given before.

The subjects of the lectures include: The first lecture; "The Fundamentals of Nutrition;" "A Balanced Ration": Second Lecture; "The Normal Diet", "The 'Alkaline Diet' Explained"; "Foods that produce 'Acidosis', and Foods that help to correct it": Third Lecture, "The Secret of Life" or "The Life-Secrets of Foods" (Vitamins) "Why Colitis?" Fourth Lecture, "21 'Varieties' of Sour Stomach and How to Avoid Them": "Ulcer and Cancer of the Stomach": Fifth Lecture, "Constipation the 'Mother' of Disease": How to overcome and live above it without Laxatives or treatment: Sixth Lecture, "High Blood Pressure": Why have it? How prevent it? How reduce it? "Heart Disease": "Bright's Disease": Seventh Lecture, "Protein the Muscle-Builder": Where to get the best and how much, and the harm of too much; Remarkable endurance records; Eighth Lecture, "Flirting With Death": "The Most Popular Sin Against Heart, Kidney, Lungs, and Nerves: A Common Cause of Asthma": Ninth Lecture, "The Chain that Binds 95% of the People to Ill Health" or "Aliens Undermining the Constitution" Two of the "links" in this "chain" are Alcohol and Narcotics; The concluding lecture will be on the topic "The Right Use of the Mind in the Conquest of Disease."

In conjunction with the lectures interesting exhibits will be on display, such as: "A Square Meal". a dinner for one person; "An 'Alkaline' Dinner for one person"; A display of the common foods containing all of the vitamins; A breakfast, dinner, and supper that will not ferment; A display of blood-pressure-raising foods; a display of blood-pressure-lowering foods; and finally a display of the right amount of proteins.

Diet lists to correct various ailments and conditions as well as many pages of valuable information about foods, nutrition, and special diets will be furnished free to those who attend.

TUBERCULOSIS ABSTRACTS

BY THE NATIONAL TUBERCULOSIS ASSOCIATION

OCTOBER, 1931

Collapse therapy in tuberculosis is now an established procedure of unquestioned value. Clive Riviere said: "No more hopeful ray of sunshine has ever come to illumine the dark kingdoms of disease" than artificial pneumothorax. This therapeutic procedure, however, has its limitations. Mechanical difficulties brought on by the pathological condition of the lung often make collapse by the insufflation of air or gas impossible or ineffective. Latterly, the injection of oil into the pleural cavity for certain cases has found favor, especially among French phthisiologists. Somewhat timidly, oleothorax is being introduced and practiced in this country. From an article by L. E. Oppengame in the *American Review of Tuberculosis*, June, 1931, the following abstracts are derived:

OLEOTHORAX

Oleothorax is the injection of mineral or vegetable oil into the pleural cavity. The indications for its use are: To avoid adhesions, to produce a more effective collapse when pneumothorax is ineffective, to give more rigidity to the pleura if a bulging of the mediastinum occurs, to close a perforation of the lung, to change an empyema into a sterile disinfectant oil. In rare cases, it may be used instead of artificial pneumothorax when for any reason a patient cannot return for routine refills.

According to Fontaine's statistics, about 70 per cent. of pneumothorax treatments are complicated by pleurisies, 22 per cent. result in empyema,

and 15 per cent. make further collapse therapy impossible because of resulting adhesions. The reason pneumothorax continues to be used in the face of these troublesome sequelae is because not all pleurisies are intoxicating for patients and a great number are beneficial for pulmonary tuberculosis. In fact, by some specialists, the development of pleural effusion is regarded as a natural healing phenomenon. There is evidence to show that pleural fluid has a deterrent influence on the development and the virulence of the tubercle bacillus. But certainly not all pleurisies are benign. Dumarest has classified pleurisies into the following groups:

Tolerated Pleurisies with little, torpid, precocious effusion produced by trauma or resulting from irritation of the pleural membranes from air insufflation. In these cases, there is no fever or pain, and reabsorption sometimes occurs by itself.

Acute Febrile Effusions (*serofibrinous*) which may appear any time during pneumothorax treatment but frequently between the third and fifth months. Effusions are profuse, there are chills, fever, a stitch in the affected side, dyspnea, irregularity of pulse, increased expectoration, and cough. The condition passes through phases but may persist for months. It is mostly benign, often regresses, and usually disappears when air refills are discontinued, but may subsequently cause adhesions. Empyema may, however, develop from it.

For these two forms of pleurisy, it is often better, after the acute stage is over, to aspirate the fluid and refill with air.

Malignant Pleurisies, or purulent, chronic effusions with high and prolonged fever, and causing malnutrition, may develop from the above-mentioned forms. The difference is not easily recognized at once; only the evolution of the disease enables one to make the differentiation. Such malignant pleurisies result in complete prostration or amyloid degeneration and usually end fatally.

With rare exceptions, there is no use for oleothorax in benign pleurisies, but in malignant cases, the first step in any intervention should be an oleothorax. Even if the operation is unsuccessful, the patient is improved and thereby placed in a more favorable condition for further surgical interference, such as rib resection or thoracoplasty. For an empyema complicating an artificial pneumothorax (which means pleurisy with tuberculous lesions), oleothorax is the only choice as thoracotomy is almost always fatal. Thoracoplasty done for tuberculous pleurisies in general leaves a persistent fistula; oleothorax, if properly done, is entirely harmless. For secondarily infected pleurisies (streptococcic, pneumococcic), drainage and not oleothorax is the proper treatment.

Oleothorax in Lung Perforations

The use of oleothorax for lung perforations is very limited. A small perforation as the result of the breaking of adhesions or a rupture into the pleural cavity of a small, subcortical vomica not connected with a bronchus, justifies an oleothorax. Unfortunately, not very often are we able to differentiate the small, benign perforation and the large, fatal one. (The author describes differential signs.)

Temporary perforations do not need an oleothorax. Valvular perforations, not helped by deflation, are benefited by oleothorax. Large perforations are mostly fatal, but if unilateral and recognized at once, thoracoplasty is a help. Burnand and Roussel, however, injected oil in such cases, thereby disinfecting the pleura and placing the patient in better condition for a final thoracoplasty.

Used in Adhesions

With oleothorax, adhesions may be avoided or prevented, though not all adhesions need an immediate oleothorax. (The beginning of adhesions can be foreseen by manometric readings and fluoroscopy.) In unsuccessful pneumothorax, for instance, in partial pneumothorax, when only one part is collapsed far from a cavitation which remains open, air insufflations are not only useless but also dangerous because of the necessary high pressure.

Other Indications

Mediastinal hernia or bulging produced by low pressure on the opposite side of a pneumothorax may be reduced by oleothorax. Finally, in exceptional cases, if patients are not able to stay in the hospital or to return for refills, a pneumothorax may be changed into an oleothorax, which requires refills only about every two months.

Technique and Results

Accidents have occurred from the injection of badly or anciently prepared, not-neutralized vegetable oil. But if all precautions are taken in its preparation, there is no great difference in the choice of the kind of oil used. The power of reabsorption depends more on the individual than on the kind of oil. The vegetable oil is milder for the pleura and not so irritant as mineral oil. The author uses mineral oil when he wishes to avoid thickening of the pleura and to produce more compression, while the vegetable oil is used mostly in empyemata.

For the last few years, he has treated many patients with oleothorax and has not had one death directly from the operation. Reported cases of death are probably due to neglect of the pressure which oil produces—a most important factor.

Of the four case histories cited by the author, the following is quoted:

Case 2: Miss A. B., aged 21, admitted July 29, 1929. Diagnosis: Chronic pulmonary tuberculosis. X-ray Report: Bronchopneumonic type of pulmonary tuberculosis, mainly unilateral. Left Lung: Diffuse involvement of entire lung with multiple cavities in upper lobe. Right Lung: Negative, except for some hazy appearance at apex and increased size of hilar lymph nodes (?); lower bronchial roots somewhat prominent.

January 30, 1930: Initial pneumothorax (400

cc.,-4, -1). February 20, 1930: After several refills reëxamination of the chest shows left-sided hydropneumothorax, with fluid level up to third interspace and displacement of mediastinum and trachea to the right. In the region of the left upper lobe is seen some lung tissue, with well-defined and sharply demarcated areas of lessened density which are undoubtedly uncollapsed vomicae held by adhesions to the chest-wall.

February 24, 1930: Lemon-colored fluid aspirated from left chest (500 cc.); 30 cc. of oil-gomenol injected; normal reaction. Patient had in four- to six-day intervals several injections until complete blockage was obtained by June 30, 1930. Since the last injection, we have controlled the amount of oil once every four weeks. The reabsorption of oil in this case since a blockage was obtained was very slow, and we injected only 5 to 10 cc. of oil once a month.

This case is particularly interesting. Why did we change an acute febrile effusion into an oleothorax and what effect was obtained by an oleothorax? Because in spite of the effusion, the cavitations in the upper lobe were open on account of adhesions and because a menacing mediastinal hernia (bulging) occurred which interfered with the further pneumothorax insufflation. After 2-3 months of oil treatments, we obtained a rigidity of the mediastinal pleura, the hernia disappeared, and we were able to inject a considerable amount of oil without fear of rupture. In the meantime, the patient developed after a pneumonia a right-sided pulmonary lesion. We feared to perform a right-sided pneumothorax on account of the left-sided oleothorax, and decided to inject sanocrysin intravenously. The patient is now in a very good condition. — *Oleothorax, L. E. Oppengame, Amer. Rev. of Tuberc.*, June 1931.

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

Bright's Disease*

A radio talk on Bright's Disease has been requested. In 1827, Dr. Richard Bright, a great London physician, clearly pointed out that swelling of the face, arms, and legs, associated with the presence of albuminuria, was due to acute disease of the kidney. Hence the term Bright's disease. But Dr. Bright described only one form of kidney disease, that with swelling of the subcutaneous tissues, or edema, as it is called. This term, Bright's Disease, is gradually becoming obsolete, and it is well that it should, for it is employed, not for a single disease, but embraces both acute and chronic generalized inflammations of the kidneys. A better term for this group of diseases is nephritis. To the average layman, the word Bright's disease has an ominous sound. But as a matter of fact, the majority of cases of nephritis are mild.

For the purposes of this talk, Bright's disease may be divided into its two commonest forms: First, acute nephritis or acute Bright's disease; and second, chronic nephritis, or chronic Bright's disease.

In brief, acute nephritis or acute Bright's disease, is a disease of the kidneys, appearing usually in the presence of some acute infection of the body, and from which the vast majority of patients recover.

Acute nephritis is chiefly a disease of children and young adults, because the acute infections are more common in the young. The most common infections that cause acute Bright's disease are scarlet fever and tonsillitis, both of which are usually due to the germ called the streptococcus. The relation between exposure to cold and acute Bright's disease seems definite in many cases, but in these it is probable that the chilling merely lowers bodily resistance and permits the germs to multiply, and the poisons they form produce the disease. Although most of the diseases that cause acute nephritis have the symptom sore-throat or tonsillitis, this does not mean that the indiscriminate removal of healthy tonsils will prevent acute Bright's disease.

Since any acute infections may cause acute Bright's disease, the only sensible and safe plan is to have all children with acute illnesses under the care of a physician who will often detect acute Bright's disease even when the patient appears and feels well. Since the only evidence of the disease may often be found in the urine, the disease could easily be overlooked. In cases of scarlet fever, the disease may not, or usually does not, appear till the third week, when the rash has already faded, and the patient may seem to his parents to have entirely recovered.

Most commonly the history is that following some acute infection the patient notices that his urine is tinged, or even dark red, or that it has a smoky color, or that he passes less urine than normal, although more frequently; or he notices that his face, hands, or legs are swollen. Other than these symptoms, however, there is nothing else that is characteristic. Pain in the back is a very uncommon symptom of acute Bright's disease, in spite of a widespread belief.

In the treatment of acute Bright's disease, the physician keeps two facts in mind; 1, that from some point in the body, often the tonsils, toxins or poisons have been formed; and 2, that the kidneys are injured. Usually, however, the acute infection such as scarlet fever has subsided by the time the kidneys are damaged, so that it is too late to pay attention to the "match that set the fire".

The immediate need is to rest the damaged kidneys. The patient should be kept in bed, warmly clothed, and with plenty of fresh air. This often means many weeks in bed—from a minimum of one month, up to six months or more, until all signs of kidney irritation have disappeared. It is

*WBZA radio broadcast September 10, 1931 at 5.17 P. M.

here that the physician requires the coöperation of the parents, who may not understand the need of keeping in bed a sick patient who may look and feel well. There is another way of resting the kidneys, and that is by diet. Since the normal work of the kidneys is to remove from the blood any excess of salt, water, and certain waste products, the physician prescribes a diet which has the minimum of these products. As a rule the patient is instructed to drink no more than six glasses of fluid a day. If there is marked swelling of the body, the drinking of fluids must be limited more strictly. However, food containing sufficient energy, vitamins, and proteins must be supplied. Since the human skin and bowels are able to take over some of the work of the kidneys, the skin and bowels are cared for daily by a tepid bath on the one hand, and mild laxatives on the other. There are no drugs that have any curative value in acute Bright's disease.

The great majority of these cases recover; a certain probably small number become chronic, and a few go on to coma and death.

Whereas acute Bright's disease is chiefly a disease of the young, the most common variety of chronic Bright's disease occurs chiefly during middle life. The great majority of cases of chronic Bright's disease are associated with high blood pressure, but it must be clearly understood that only a small percentage of the large number of people with high blood pressure ever develop chronic Bright's disease or chronic nephritis. Since high blood pressure tends to run in families, chronic Bright's disease therefore frequently tends to run in families.

It has not been shown that eating certain food, for example, meat, or eating too much food in general, is the cause of this disease in the human being. The Eskimos live on a meat diet, yet Bright's disease is no more frequent than it is among people who eat little meat. There is, therefore, no sound reason for healthy people abstaining from any special foods in attempting to prevent this disease. The same applies to people with simple high blood pressure.

Chronic Bright's disease is an insidious disease. Many people who feel in the best of health or who have only slight complaints may already have advanced forms of the disease. It is often discovered at some periodic health examination. However, many patients with this disease complain of many symptoms such as headache, irritability, weakness, loss of weight, and frequent passing of urine, especially at night. None of these symptoms, however, are characteristic of the disease.

On examination such patients may show absolutely nothing except changes in the urine such as albumin, etc. Most commonly, however, patients with chronic Bright's disease are found to have a high blood pressure.

The treatment of chronic Bright's disease consists in general hygiene and diet. Nothing is known that will cure the disease. The most important thing to do is to live moderately in all ways—moderate physical and mental exertion, moderate eating, drinking and tobacco. Since coffee, tea, alcohol, salt, and proteins give the kidney more work, a diet is usually prescribed that limits all of these. There is no difference chemically between red and white meat. Beef is the same as chicken. It must be emphasized that a varied and abundant diet is necessary in a disease that is chronic. The doctor will decide by his examinations how much meat may be taken. The amount of water to be taken is not limited, but rather is given in abundance. The heart should be protected by adequate rest and by keeping the weight down to the normal.

In the treatment of this most common form of chronic Bright's disease, which is associated with high blood pressure, it is important to realize that we are dealing as much with a certain type of individual as with a certain disease. People who develop high blood pressure and this form of chronic Bright's disease, usually are hyperactive individuals, either physically or mentally, or both. Very often they lose their temper easily, or are hypersensitive. Just as often they are the type of person who is always "on the go", working rapidly, and living a life of continual motion. In the treatment, the attempt to modify these abnormal reactions is just as important as diet. In other words, mental and physical relaxation are very important, as well as the development of mental serenity.

The course of chronic Bright's disease varies. Many people live for many years after the discovery of the disease. A life of moderation under the observation of a physician undoubtedly prolongs the life of such patients.

The Massachusetts Medical Society

TO THE SECRETARIES OF THE DISTRICT MEDICAL SOCIETIES

American Medical Association
Bureau of Medical Economics
535 North Dearborn Street, Chicago
September 17, 1931.

Dr. W. L. Burrage, Secretary,
Massachusetts Medical Society,
Brookline, Mass.

Dear Sir:

The Bureau of Medical Economics, which has been established to study all phases of general economics having a bearing on the practice of medicine, is now prepared to initiate several of its studies. A fair summary or analysis of the several phases of medical economics must, of necessity, take into consideration conditions, practices, tendencies and opinions as they actually exist throughout the country.

In order that we may obtain the most reliable information upon which to formulate general policies

and principles, we are asking the state and county medical societies for definite information concerning those phases of medical economics with which they have had experience.

Every phase of medical economics upon which information is sought in these schedules is of great importance to the medical profession. We urge that you do not lay the schedules aside. The answers to the questions asked may be helpful to your society, especially if it becomes necessary for you to make some investigations to enable you to answer the questions completely. It is essential that we have your reply before we shall be able to formulate any opinion on the subject for the entire United States.

We assume that it may require a little time to formulate answers to some of the questions; however, we shall appreciate as early a reply as may be consistent with thoroughness and comprehensiveness.

Yours very sincerely,
R. G. LELAND, M.D., *Director*,
Bureau of Medical Economics.

SCHEDULE OF MEDICAL ECONOMICS

Date.....193....

To: THE BUREAU OF MEDICAL ECONOMICS, AMERICAN MEDICAL ASSOCIATION, CHICAGO, ILLINOIS.

.....Medical Society, with headquarters at
City.....State.....
President.....Address.....
Secretary.....Address.....
Regular Meeting Day.....Number of Members.....

K. SICKNESS INSURANCE

1a. Is there any sentiment in your medical society in favor of compulsory health insurance? YES ☐ NO ☐.
1b. In favor of voluntary health insurance? YES ☐ NO ☐.
2a. Are there any organizations (lay, labor, insurance or other) in your community seeking to create sentiment in favor of compulsory health insurance? YES ☐ NO ☐.
2b. In favor of voluntary health insurance? YES ☐ NO ☐.
3. List such organizations here by name and address.
4a. What arguments have been advanced in your community in favor of compulsory health insurance?
4b. In favor of voluntary health insurance?
5a. Is the sentiment in your society unfavorable to compulsory health insurance? YES ☐ NO ☐.
5b. To voluntary health insurance? YES ☐ NO ☐.
6a. What arguments are advanced by your society against compulsory health insurance?
6b. Against voluntary health insurance?

J. MEDICAL FEES

1. Has your county medical society adopted a fee schedule? YES ☐ NO ☐.
2. How recently have these schedules been revised?
3. Attach here a copy of the county medical society fee schedule.
4. Do members of the county medical society adhere to the present fee schedule? YES ☐ NO ☐.
5. If not, what explanation can you give for departing from this schedule?

H. INDUSTRIAL MEDICINE

1. Are the present arrangements with respect to dealings between the State Department of Industrial Relations or the State Industrial Commission and the medical profession on workmen's compensation cases satisfactory? YES ☐ NO ☐.
2. If not satisfactory, how could the relations be improved?
3. Did the State Medical Association take any part in forming the state workmen's compensation law? YES ☐ NO ☐.
4. In what other ways has the State Medical Association been actively interested in the state workmen's compensation law?
5. Attach here a copy of the industrial accident and occupational diseases fee schedule.
6. Is this schedule satisfactory to the State Medical Association? YES ☐ NO ☐.
7. If not satisfactory indicate here the changes that should be made.
8. Are physicians' claims for the care of industrial accidents and occupational diseases settled promptly and fully by your State Department of Industrial Relations? YES ☐ NO ☐.
9. If not settled promptly and in full what are the reasons for delay in adjustment?

CORRESPONDENCE

"VULNERABLE HYPOTHESES" RESPECTING ANTERIOR POLIOMYELITIS

City of Newton, Massachusetts
City Hall
West Newton

September 21, 1931.

Editor of the *New England Journal of Medicine*,
Sir:

Dr. Delano's communication on poliomyelitis in the *Journal* of August 27, seems to me to be very timely and worthy of careful consideration.

To those of us who recall the occurrence of polio in the past, I think that there can be no doubt that the answer to his question as to whether polio is not somewhat snarled up, should be in the affirmative.

Many of us who remember the time when patients with polio were treated in a general hospital with no precautions against transmission from person to person, and without any such transmission,

are somewhat confused and, perhaps, a bit sceptical when we are confronted with modern hypothesis in regard to the disease and its occurrence.

We are told that it is communicable from person to person; that many persons have had it without knowing it and are therefore immune; and that it may be transmitted by healthy carriers, who never knew they had it.

These last two hypotheses are, of course, necessary to explain the infrequency of multiple cases in a family and the occurrence of cases in localities where the disease is not present and there has been no demonstrable direct, or indirect, exposure to known cases.

These hypotheses are rather difficult to combat but it seems to me that they are more or less vulnerable.

Take the question of communicability:—Every health officer knows that the occurrence of multiple cases in a family is extremely rare, except in those instances in which the cases develop in such a manner that it may be said that they may have and probably did, acquire their infection at the same time from some common source but, because of a varying resistance, the appearance of the symptoms was delayed in individuals. These multiple cases do not develop in sequence after the lapse of a period of incubation, as is the case in other communicable diseases where the second case receives its infection from the first one and so on.

Again we are all familiar with instances in which one child, in a family of several, develops polio and no other case follows even though no precautions against transmission are taken. This is so frequent that it may almost be looked upon as the rule rather than the exception.

As an extreme example I may cite an instance where one child in a family of seven developed polio and remained in the most intimate contact with the other children, even sleeping in the same bed, for a week after the paralysis had appeared, before the case was discovered and yet no other case occurred in that family or even in the immediate neighborhood. This is not an isolated example as anyone used to dealing with polio can testify.

Imagine what would have happened had the disease been measles, scarlet fever, whooping cough or any other of the recognized communicable diseases.

It seems too much to ask us to believe that the other children, not only in the family but in the neighborhood, had had polio, some time in the past, without knowing it and had developed an immunity which protected them.

Finally as to the healthy carrier who is held responsible, in many instances, for the spread of infection. It seems to me that he is more sinned against than sinning.

I do not overlook the part played by the carrier in spreading infection, but it seems to me very curious that he should be able to spread infection, when frank, open cases, even when unisolated:—cases in which there can be no question of the diagnosis, seem unable to do so.

I cannot recall any other communicable disease in which this peculiar condition exists.

I think that there is a tendency to go ahead too fast and that many of the hypotheses in regard to polio are based upon assumption rather than fact.

The conclusion seems almost inevitable that polio is not communicable from person to person, and from this it follows that the almost hysterical fear of persons who have polio is entirely unjustified.

FRANCIS GEO. CURTIS.

DR. WOODWARD WOULD LIKE TO BE INFORMED OF THE ATTITUDE OF STATE SENATORS TOWARD THE COMPULSORY VACCINATION BILL

Mr. Editor,

I have no desire to clutter up your columns with vaccination matter at this time but would be grateful for any information which would give me an idea of the attitude of individual State Senators toward my bill to extend compulsory vaccination to the private schools. Last year the unanimous vote in the Senate was to reject it, a rather disconcerting action particularly after approval by the House by a 3 to 1 vote.

Sincerely yours,

SAMUEL B. WOODWARD.

58 Pearl Street, Worcester, Mass.

RECENT DEATH

KEARNEY—DR. JOSEPH PATRICK KEARNEY, chief district physician for the City of Lowell, died in that city September 25, 1931, aged 51. Born in Lowell, he was educated in the Lowell schools and at Ottawa University. His medical degree was from Tufts College Medical School in 1908. The following year he joined the Massachusetts Medical Society. Dr. Kearney saw service in the World War and was a member of the Lowell Post, American Legion, also of the Lowell Order of Eagles, the Nashua Country Club and the Longmeadow Golf Club. He had been local examiner for the John Hancock Life Insurance Company. Kearney Square in Lowell was renamed for Dr. Kearney's brother, Paul T. Kearney, who was killed in action during the War.

OBITUARY

RESOLUTIONS ON THE DEATH OF DR. WILLIAM A. SHEEHAN

William A. Sheehan, M.D., Associate Anesthetist to the Carney Hospital, died at the home of his parents in Boston, May 18, 1931.

Dr. Sheehan was a graduate of the Boston University School of Medicine in 1926, and chose the Administration of Anesthetics as his specialty. In this work he became very proficient and skilful. Quiet, unpretentious in manner, observing and

ever ready to improve himself; kindly and painstaking with his patients, Dr. Sheehan developed for himself an enviable reputation among his associates and greatly endeared himself to his patients.

The Carney Hospital realizes that it has suffered a loss, and it is the wish of the Governing Board and the Staff that this expression of sorrow and sympathy be sent to Doctor Sheehan's family and spread on the official records of the Governing Board of this Institution.

DANIEL F. MAHONEY,
Secretary of the Governing Board.
Carney Hospital, South Boston, Mass.
September 21, 1931.

NEWS ITEMS

A MEMORIAL TO RONALD ROSS—Robert L. Pitfield, M.D., Secretary of the Ross Award Fund of America, has issued an appeal for contributions to the Ross Memorial.

Ronald Ross, I.M.S., in 1896 discovered the manner in which malaria is conveyed by mosquitoes.

The address of the Ross Award Fund is 5211 Wayne Avenue, Germantown, Pa.

HARVARD MEDICAL SCHOOL NOTES

The Department of Anatomy of Harvard Medical School has been reorganized with the appointment of three full professors of anatomy. Dr. Frederic T. Lewis, formerly Associate Professor of Embryology, has been appointed to the James Stillman Professorship of Anatomy, a chair last occupied by Charles Sedgwick Minot (1905-1914). Dr. J. Lewis Bremer, formerly Associate Professor of Histology, is now the Hersey Professor of Anatomy, a position last held by Edward Laurens Mack (1885-1921).

Dr. George B. Wislocki has come from Johns Hopkins to become the Parkman Professor of Anatomy. The late Thomas Dwight held this position from 1883 to 1911. Dr. Wislocki was graduated from Washington University (St. Louis) in 1912 and from Johns Hopkins Medical School in 1916. He was an assistant in anatomy in that school during the following year and served in the United States Medical Corps during 1918. From 1918 to 1920 he was the Cabot Fellow in charge of the laboratory for surgical research at Harvard Medical School. He then returned to the anatomic department at Johns Hopkins and was appointed an associate professor in 1923. His chief interest has been in anatomic and physiologic research concerning reproduction and he has published a number of papers in this field.

Dr. David M. Rioch (Johns Hopkins 1924) is also coming to Harvard from Johns Hopkins as an associate professor of anatomy in charge of neurology and Harold L. Weatherford, Ph.D., has been advanced from instructor in anatomy to associate professor in anatomy.

NOTICES

RADIO HEALTH MESSAGES

Sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

Courtesy WBZA. Thursdays at 5:17 P. M.

October

8 What a Health Examination Means
15 Sunlight and Ultra-violet Rays
22 Hospitals—Then and Now
29 What the Public Should Know of Diabetes

November

5 High Blood Pressure
12 The Care of Pulmonary Tuberculosis
19 Accidents in the Home
26 Fatigue as a Symptom of Disease

December

3 Overweight—Cause and Prevention
10 Can We Prolong Life?
17 Pasteurized or Raw Milk
24 Public Health in the Coming Year
31 Mental Health

STATE HOUSE BROADCAST

Sponsored by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays at 12:30 P. M.

The various activities of the Department are discussed by the Directors of its nine Divisions.

RADIO HEALTH FORUM

Directed by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays at 4:50 P. M.

Queries from the public are answered under the sponsorship of the Department.

Questions on Health and Prevention of Disease may be sent to: Radio Health Forum, State Department of Public Health, State House, Boston.

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3.30 P. M. on Thursday, October 8, in the Amphitheatre of the Peter Bent Brigham Hospital, a medical clinic will be given by Dr. Henry A. Christian, to which physicians are cordially invited. The clinics will be held regularly on Thursday afternoons.

REPORTS AND NOTICES OF MEETINGS

BRISTOL COUNTY ORTHOPAEDIC CLUB

The Bristol County Orthopaedic Club held a Clinical Meeting at the Truesdale Hospital, 1820 Highland Avenue, Fall River, Mass., on Tuesday evening, September 29, 1931, at 8:30 P. M.

Dr. Clarence C. McCreery presented the following cases:

A. Pathology of the Femur Bone—Undiagnosed.

B. Reconstruction Operation—Old Fracture of the Femur.

C. Derangement of the Cartilages of the Knee Joint.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

At a meeting of the executive committee the following schedule for the Middlesex East District Medical Society was arranged:

November at Wakefield,
January at Melrose,
March at Reading,
May at Woburn.

All meetings on the second Wednesday of each month unless otherwise announced at a later date.

At the September meeting Dr. A. Warren Stearns delivered a very interesting and instructive address.

MIDDLESEX SOUTH DISTRICT MEDICAL GOLF ASSOCIATION

The Fall tournament of the Middlesex South District Medical Golf Association was held on Wednesday, September 16, at the Belmont Spring Country Club, Waverley, Mass.

The low gross of the tournament was won by Dr. Charles Liebman with an 82. Dr. C. T. Porter was second gross and Dr. William T. O'Halloran third gross.

Dr. Bert Macchia won low net, Dr. M. E. Cummings second net and Dr. A. J. Campbell third net. Dr. M. Ritvo captured high gross, Dr. Frank Gerry, low gross for nine holes and Dr. W. S. Whittemore high gross for nine holes.

The attendance was gratifying to the officers and indicative of the growing popularity of this event.

Due to an unfortunate oversight another event was scheduled on the same day and resulted in considerable congestion at times. This prevented several of our Fellows from playing. Fifty-seven, however, turned in scores and reported a good time. Twenty-five stayed for dinner, at which time the prizes were presented.

Watch the *Journal* for notice of the Spring Tournament next June.

S. M. Biddle, *Secretary-Treasurer.*

SOUTH END MEDICAL CLUB

The opening meeting of the South End Medical Club will be held on Tuesday, October 20, 1931, at 12 noon, at the offices of the Boston Tuberculosis Association, 554 Columbus avenue, Boston. The speaker will be Arthur T. Legg, M.D., Associate Orthopedic Surgeon, Children's Hospital, and his subject, "The Early Treatment of Infantile Paralysis." Lantern slides will be used.

All physicians are cordially invited to attend. Luncheon will be served at 1 o'clock.

JOINT MEETING OF THE GREATER BOSTON MEDICAL SOCIETY AND THE BETH ISRAEL HOSPITAL

There will be a joint meeting of the Greater Boston Medical Society and the Beth Israel Hospital on Tuesday, October 6, 1931, at 8:15 P. M. at the Beth Israel Hospital.

PROGRAM

Intracranial Dynamics
Abraham Myerson, M.D.
The Present Status of Poliomyelitis
Sidney D. Kramer, M.D., Harvard Infantile Paralysis Commission.
Discussion from the floor
Bernard I. Goldberg, M.D.

SOCIETY MEETINGS

CONGRESSES AND CONFERENCES

October 5, 6, 7, 8—American Congress of Physical Therapy. See notice on page 272, issue of July 30.

October 6—Joint meeting of the Greater Boston Medical Society and the Beth Israel Hospital. See notice above.

October 8—Medical Clinic at the Peter Bent Brigham Hospital. See page 701.

October 8-9—Vermont State Medical Society. See page 493, issue of September 3.

October 12-16—Announcement of the Clinical Congress of the American College of Surgeons. See page 1175, issue of May 28.

October 12-16—Special Course in Anesthesia. See page 555, issue of September 10.

October 14—The New England Dermatological Society. See notice on page 655, issue of September 24.

October 19-23—International Assembly of the Interstate Postgraduate Medical Association of North America. See page 1125, issue of May 21, and page 555, issue of September 10.

October 20—South End Medical Club. See notice elsewhere on this page.

October 19-30—Graduate Fortnight of the New York Academy of Medicine. See page 1230, issue of June 4, and page 465, issue of August 27.

April 4-8, 1932—The American College of Physicians. See notice on page 1232, issue of June 4.

Autumn 1931—Spring 1932—International Medical Postgraduate Courses in Berlin. See page 468, issue of August 27.

DISTRICT MEDICAL SOCIETIES

Franklin District Medical Society

Meetings will be held on the second Tuesday of November, January, March and May at the Weldon, Greenfield, at 11 A. M.

CHARLES MOLINE, Secretary.

Middlesex East District Medical Society

November-May—See notice elsewhere on this page.

Worcester District Medical Society

Wednesday evening, October 14, 1931—Worcester Chamber of Commerce Rooms, Franklin Street, Worcester, Mass. 7:45—Program: Recent Advances in the Etiology and Treatment of Cancer, by Dr. Clarence C. Little, Bar Harbor, Me.

Wednesday evening, November 11, 1931—Worcester Chamber of Commerce Rooms, Franklin Street, Worcester, Mass. 7:45—Program: Bone Lesions of Congenital Syphilis, by Dr. Stafford McLean, Clinical Professor, Diseases of Children, Columbia University College of Physicians and Surgeons, New York City.

Wednesday evening, December 9, 1931—Worcester Chamber of Commerce Rooms, Franklin Street, Worcester, Mass. 7:45—Program: Subject in the field of Eye, Ear, Nose and Throat to be announced later.

Wednesday evening, January 13, 1932—Worcester City Hospital, Worcester, Mass. 6:30—Dinner, complimentary by City Hospital. 7:45—Program: To be announced later.

Wednesday evening, February 10, 1932—Worcester State Hospital. 6:30—Dinner, complimentary by the Worcester State Hospital. 7:45—A psychiatric program to be announced later.

Wednesday evening, March 9, 1932—Memorial Hospital, Worcester, Mass. 6:30—Dinner, complimentary by Memorial Hospital. 7:45—A pediatric meeting with subjects to be announced later.

Wednesday evening, April 13, 1932—Grafton State Hospital, North Grafton, Mass. 6:30—Dinner, complimentary by Grafton State Hospital. 7:45—Program to be announced later.

Wednesday afternoon and evening, May 11, 1932—Annual Meeting. Program to be announced later.

ERWIN C. MILLER, Secretary.